THE American Journal OF Gastroenterology

VOL. 24, NO. 2 AUGUST, 1955

The Recognition and Present Treatment of Endemic Fish Tapeworm Infestation (Diphyllobothriasis)

Comprehensive Testing of Gastric Secretory Function

A Consideration of Gastroduodenal Disorders

Volvulus of the Newborn

The Pancreatic Secretin Test as a Diagnostic Aid

Complete Healing of Peptic Ulcer with Water Soluble Chlorophyll

Second Annual Convention
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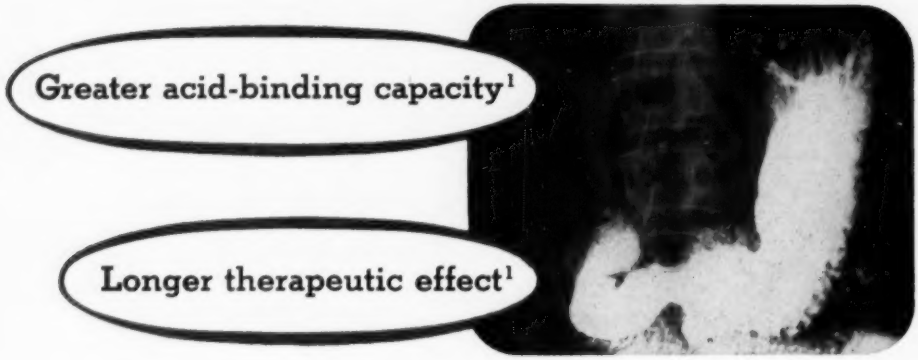

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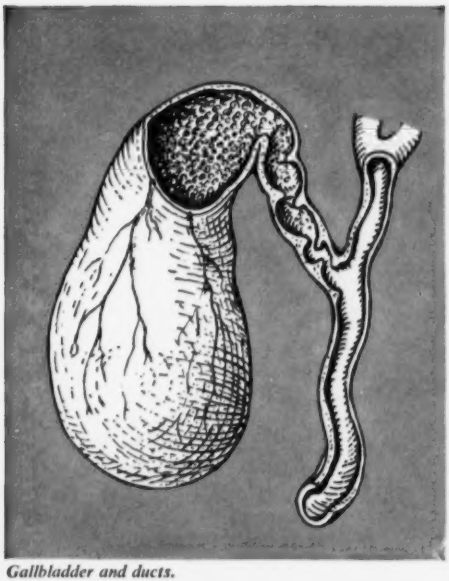

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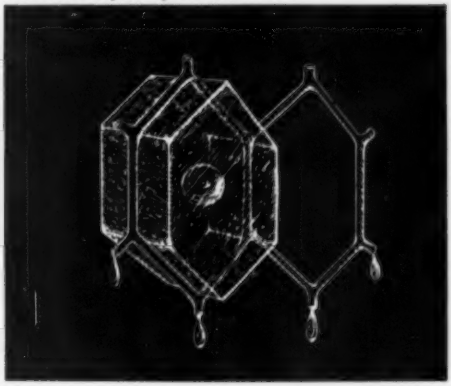

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The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

contents:

Editorial Board and General Information 116

The Recognition and Present Treatment of Endemic Fish Tapeworm Infestation (Diphyllobothriasis) ..... JULIUS ROSENBERG, M.D., EDITH NEUMANN, Ph.D. and MILTON MATZNER, M.D., F.A.C.P., F.A.C.G. 121

Comprehensive Testing of Gastric Secretory Function
GEORGE B. JERZY GLASS, M.D. and MARILYN RICH, A.B. 137

A Consideration of Gastroduodenal Disorders . . PETER G. BRANDETSAS, M.D. 153

Volvulus of the Newborn ..... ROY MORROW, M.D. and KENNETH JUDY, M.D., F.A.C.S. 167

The Pancreatic Secretin Test as a Diagnostic Aid . . . . . DAN ZAVELA, M.D., LOUIS GUZZETTA, M.D. and RUTH DAVIS MCNAIR, Ph.D. 177

Complete Healing of Peptic Ulcer with Water Soluble Chlorophyll
W. G. OFFENKRANTZ, M.D. 182

In Memoriam 185

President's Message 186

Abstracts for Gastroenterologists 187

Book Reviews for Gastroenterologists 191

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Index to Advertisers

Ames Co., Inc. 120
Ciba Pharmaceutical Products, Inc. 199
Endo Products, Inc. 195
Hoffmann-La Roche, Inc. 114
Horlicks Corp. 196
Lakeside Laboratories, Inc. 198
National Drug Co., The ..... 3rd Cover
Pfizer Laboratories 118, 119
Rorer, William H., Inc. ..... 2nd cover, 193
Searle, G. D. Co. 113
Sharp & Dohme 196
Standard Pharmaceutical Co., Inc. 192
Upjohn Co. 200
U. S. Treasury 194
Warner-Chilcott Laboratories ..... 4th cover
Winthrop-Stearns, Inc. 117
Wyeth, Inc. 197

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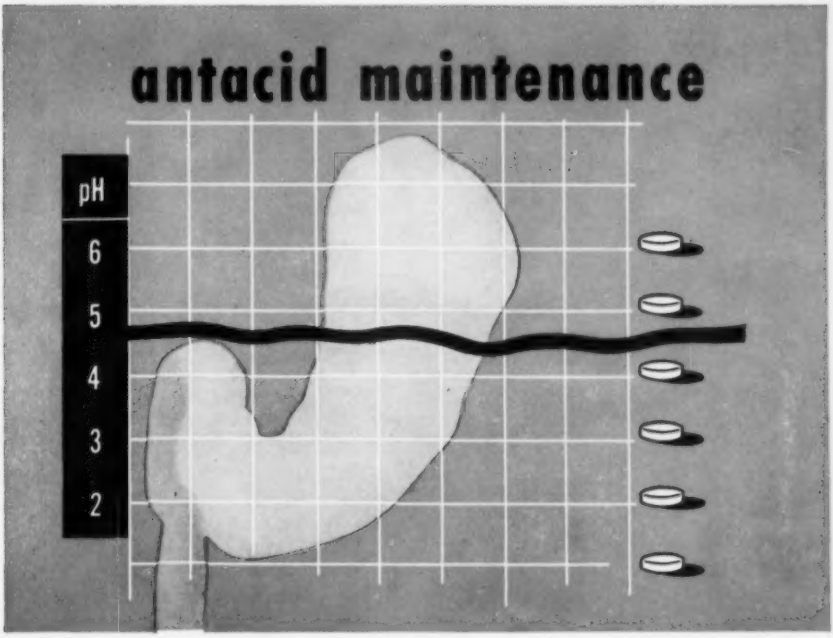

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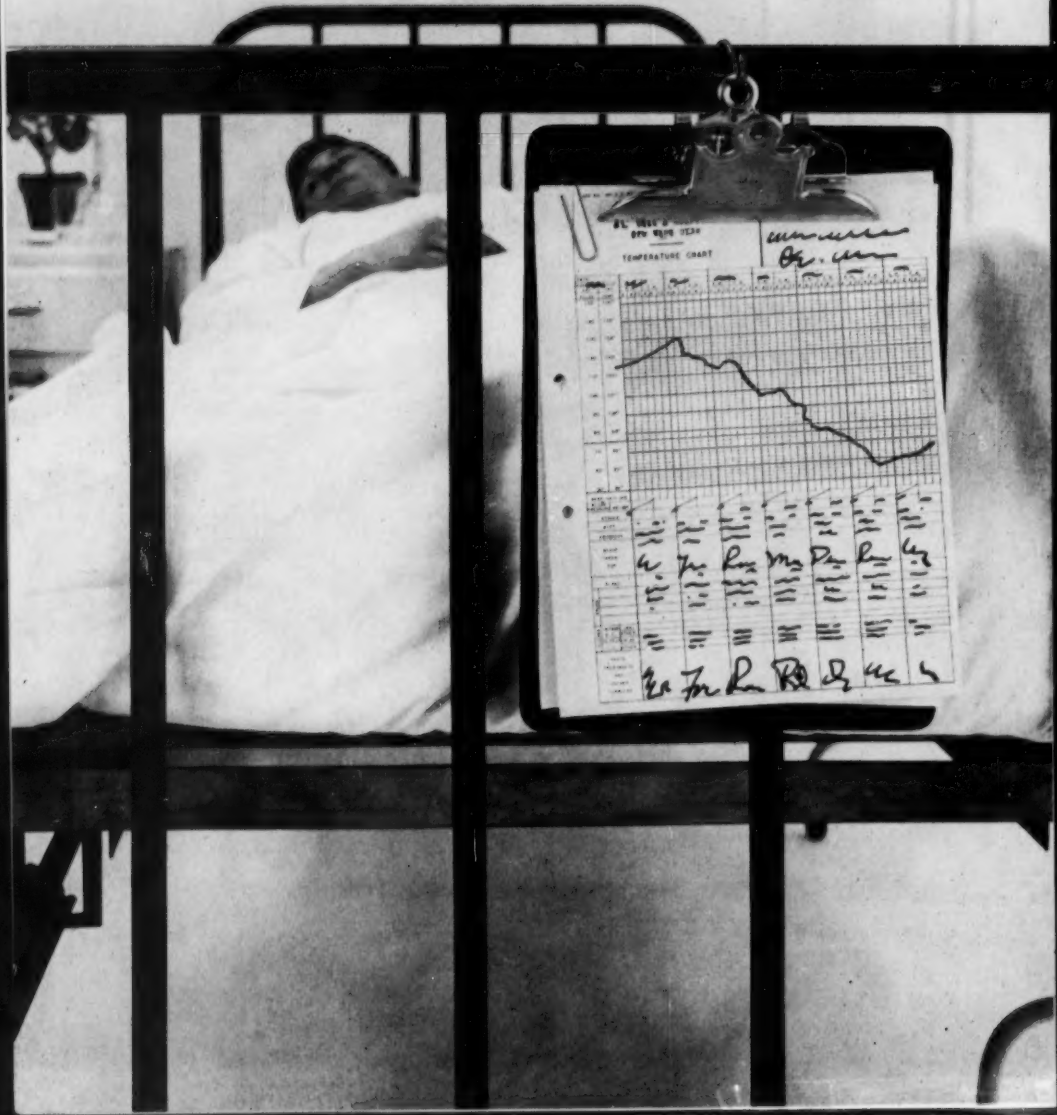

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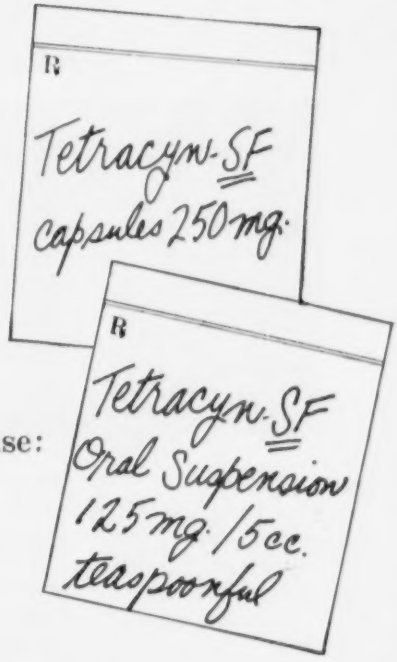

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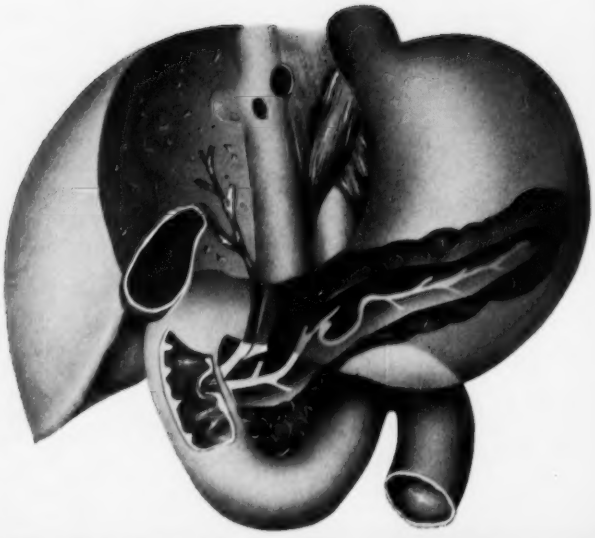

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VOLUME 24 AUGUST, 1955 NUMBER 2

THE RECOGNITION AND PRESENT TREATMENT OF ENDEMIC FISH TAPEWORM INFESTATION (DIPHYLLOBOTHRIASIS)*

JULIUS ROSENBERG, M.D.

EDITH NEUMANN, Ph.D.

and

MILTON J. MATZNER, M.D., F.A.C.P., F.A.C.G.

Brooklyn, N. Y.

During the years 1950 through 1953 a total of 13 cases of *Diphyllobothrium latum* infestation were diagnosed in the parasitology laboratory of the Jewish Hospital of Brooklyn. It is believed that it would be advantageous to again call the attention of the medical profession to this disease and its current concepts of therapy.

Fish tapeworm infestation was first recognized in this country in 1879[1], in an American who had emigrated from Sweden. In 1906 Nickerson[2] reported the first native-born United States case in Minnesota.

According to Magath[3,4] the primary introduction of *Diphyllobothrium latum* into the waters of North America was made by immigrants accustomed to eating raw fish, who came from the endemic centers of Europe, in particular from the Baltic States. Many of these people, particularly the Scandinavians and Finns, settled about the Great Lakes region of central North America where the smaller lakes contain suitable intermediate hosts to insure the development of the worm. It has been estimated[5] that about 80 to 100 per cent of the people in the Baltic and Scandinavian countries, where these immigrants came from, are infected with these worms and the lakes became infected by way of sewage containing human excreta.

Barron[6] in 1929 introduced the concept that fish tapeworm infestation in Jewish women occurred while sampling insufficiently cooked fish during the

*Read by title before the First Annual Convention of the American College of Gastroenterology, Washington, D. C., 25, 26, 27 October 1954.

From the Division of Gastroenterology, Department of Medicine and The Department of Microbiology, The Jewish Hospital of Brooklyn, Brooklyn, N. Y.

process of preparing "gefülte" fish. This was subsequently confirmed by many authors[1,7,8,9]. All of the 13 cases reported by us in this article were in Jewish women who had tasted "gefülte" fish while it was raw or being cooked or had tasted its soup during cooking.

Pathogenesis

Diphyllobothrium latum, the fish tapeworm or broad tapeworm, produces in man and in its other definitive hosts an infection characterized by the presence of the hermaphrodite adult worm in the intestinal lumen. The adult worm may measure from 3 to 10 meters in length and possesses between 3,000 to 4,000 proglottids or segments. The scolex (head) is small and spatulate with a pair of deep sulci. Proglottids and/or the operculate eggs, which measure 45 microns by 65 microns, are discharged in the feces of the definitive host. Eggs are evacuated periodically through the uterine pore of each functional proglottid. It has been estimated that a single worm may discharge as many as one million eggs per day[10].

If the ova get into fresh water they develop in 10 to 14 days into ciliated embryos (oncospheres) that swim around and die in a few hours unless eaten by an appropriate small fresh-water crustacean, a copepod of the genus "Cyclops" or "Diaptomus". These are the first intermediary host, and within their body cavity the embryo develops into the procercoid or first larval stage in about two weeks.

If the infected copepod is now ingested by a fresh water fish, the larva works its way through the tissues of the fish and comes to lie free between the muscle fibers, where it grows into a plerocercoid or sparganum larva, measuring up to 6 mm. in length. This is the plerocercoid or second larval stage. The fish involved in this country, and especially those sold along the eastern seaboard to Jewish housewives, are the wall-eyed and northern pike, carp and whitefish[11] which come from areas in the Great Lakes region and Canada, which are known to be endemic foci of the disease. Magath[3,12] and Ward[13] found infested fish in four lakes of Northern Minnesota, three of which drain into Canadian lakes and the fourth drains into Lake Superior. Vergeer[14-16] implicates the lakes around Winnipeg, Canada, including Lake Winnipeg, and also the lakes of Northern Michigan, including Lake Superior. Both intermediary hosts are present in the endemic lakes. The degree of infestation in these lakes depends on the population living nearby, infestation being greatest near communities of Finns, and Russian and Polish Jews. According to Magath[4] infestation of these lakes is maintained by sewage containing human excreta.

The infested fish are then eaten by man, dogs, cats, bears, and foxes and the cycle is completed (Fig. 1). Magath and Essex[3] questioned the part played by dogs in the spread of this infestation but Vergeer[15] showed that dogs in the region extending from Ely, Minnesota north into Canada were heavily

infested with *Diphyllobothrium latum* and were largely responsible for the continued infestation of fish with the larva. He also found eggs in the feces of wild bears. Ward[13] also confirmed the presence of infestation in the dog.

The plerocercoid larva develops, in the intestine of the host, into the adult worm in about 3 weeks and is then capable of discharging eggs. The adults of *Diphyllobothrium latum* have been known to survive for periods of 5 to 10 years. Most patients have one or two adult worms, although four have been

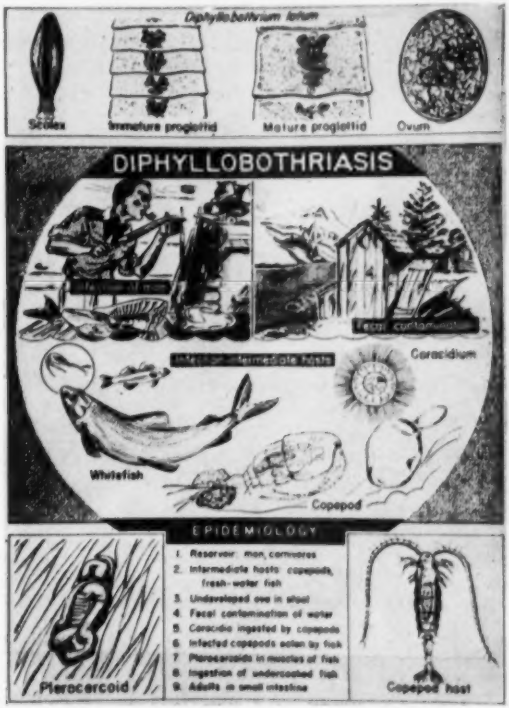

Fig. 1—Epidemiology of *Diphyllobothriasis*. From: Mackie, T. T., Hunter, G. W. and Worth, C. B., "Manual of Tropical Medicine" of The National Research Council, Second Edition, 1954, W. B. Saunders Company, Philadelphia, Pa.

reported[4]. That is why it is important to recheck the stools for ova and proglottids repeatedly after a course of therapy, and not use the finding of the head as a criterion in determining whether or not treatment has been successful.

Diphyllobothriasis has been reported, often in native-born, in New York City[7,11], Indianapolis[17], New England[9,18,19], Detroit[1], Montreal[8], and even in subtropical Florida[20] in three Negro children in whose stools ova were demonstrated in routine examinations. In two of these cases the worms were passed

after a course of treatment. There was no follow-up in the third. These children caught fish in the nearby ponds and cooked them over an open fire. The fish were probably eaten uncooked at times. The dog of one family had fish-tapeworm eggs in his stools. Cases have also been discovered in Wisconsin, Minnesota, Michigan, Oklahoma, Alabama, Ohio, Washington, D. C., Louisiana, New Mexico, Illinois, Iowa, Pennsylvania, Colorado and Texas[1]. Recently, the disease was noted in Great Britain[21] and Australia[22].

In recent years it has become apparent that most cases of fish-tapeworm infestation in this area of the country appear in Jewish housewives who have tasted "gefülte" fish or its soup prepared from infested fish before adequate cooking, or in children who tasted the raw fish during its preparation. It is not necessary to swallow the sampled food. In order to determine whether the dish is properly seasoned, Jewish housewives are in the habit of tasting the soup or particles of the "gefülte" fish and even if the housewife spits out the tasted material the viable larvae might be deposited in the oral cavity and subsequently swallowed in the saliva. All of our 13 cases were in adult Jewish females who had tasted "gefülte" fish, one as a child. Jewish men are almost never affected because the finally cooked fish is non-infectious.

The fish used in the preparation of the "gefülte" fish are pike and carp which have been shown by actual survey of wholesale fish dealers in the New York area[11] to come from the infested lakes areas mentioned above.

Among the Finnish people in this country the infestation is introduced by eating freely of various preparations of raw fish. Muntz[17] reported a case in a female who ate large quantities of raw smoked fish from the Great Lakes area. The roe as well as the flesh may be affected and some cases have been reported to have occurred from eating caviar.

The exact incidence of fish-tapeworm infestation in this country is not known since it is not a reportable disease.

Clinical Manifestations

The adult worm lives in the human intestine and the proglottids, as they ripen, break off from the tapeworm and are discharged from the intestine[19]. The adult form has lost all traces of a digestive tract and absorbs food from the intestinal canal of the host all over the surface of its body. The degree of its pathogenic effects depends in part on the mass of the worm, the type and amount of the worm's metabolic wastes absorbed into the host's tissues and the host's susceptibility to these foreign substances. In most patients there is no evidence of damage to the host.

The great majority of persons who harbor the fish tapeworm in their intestinal tracts suffer no ill effects from this parasitism[23]. Most infections either are asymptomatic or produce slight transient abdominal discomfort. The usual

story is the patient coming into the doctor's office with the history of passing a long segment of the worm and often presenting the specimen to the physician for diagnosis. This occurred in 12 out of 13 of our cases. Only six of our patients had abdominal discomfort and three of the six had other associated gastrointestinal pathology.

In some cases there will be a variety of clinical manifestations. There may be neurotic states or functional disturbances of the central nervous system[23], or there may be nonspecific gastrointestinal symptoms: functional disorders,

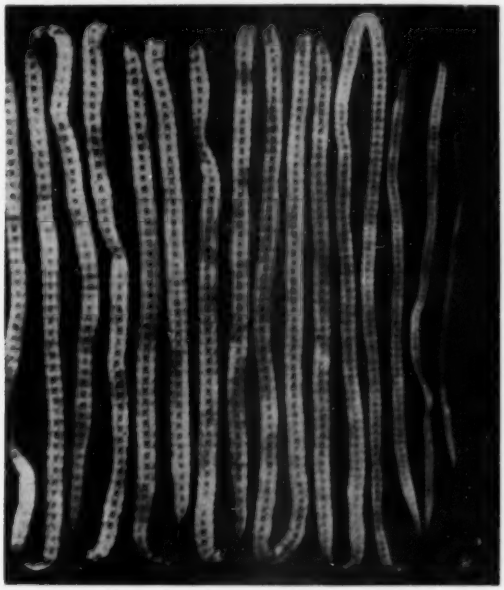

Fig. 2—*Diphyllobothrium latum*. Complete adult specimen expelled during treatment. From: Gradwohl-Kouri, Volume III, "Clinical Laboratory Methods and Diagnosis", Fourth Edition, 1948, C. V. Mosby Company, St. Louis, Mo., after Kouri and Basnuevo in "Lecciones de Parasitologia y Medicina Tropical", Volume II, (Helmintologia Humana), Second Edition, 1947.

irregularity of the bowel, stomatitis or glossitis or vomiting. There may be marked toxemia. Tarassov in 1937, after experimentally infecting himself with fish tapeworm, suffered from marked abdominal pain, lost 17½ pounds and became so weak he had to be hospitalized[5].

Von Bornsdorff[24] who has made extensive observations on fish tapeworm infestation in Finland, where its incidence is probably highest, reports that experiences from abdominal operations and autopsies show that *Diphyllobothrium latum* is most frequently found in the ileum, rarely in the jejunum and sometimes in the colon. The worm can also develop in the gallbladder. He

mentions a surgically removed gallbladder found stuffed full of worms. Fish tapeworm can also cause intestinal obstruction. According to the same observer tapeworm vomiting is a symptom of this disease that has hitherto been neglected. The attack of vomiting can be very sudden and associated with collapse, severe pains and a feeling of suffocation. Such conditions have (before vomiting up the worm) been confused with perforation of a peptic ulcer, gallstone colic, intestinal obstruction and appendicitis. Among tapeworm carriers who had vomited the worm, anemia has been diagnosed more often than among tapeworm patients in general. It is possible that in many cases the vomiting may be assumed to indicate that the worm is located in the upper part of the small intestine.

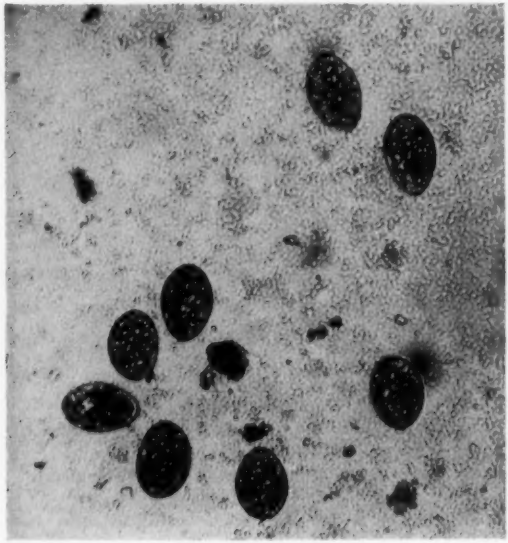

Fig. 3—*Diphyllobothrium latum.* Ova in concentration of fresh stool specimen. High power, x 500.

At times fish tapeworm infestation is associated with a severe primary megaloblastic anemia of the pernicious anemia type, usually referred to as *bothriocephalus anemia,* in which the red blood corpuscles may be reduced to one million per cubic millimeter or less. Birkeland[23] made a complete study of this subject and concluded that the actual number of cases of this anemia is very small compared with the total number of individuals infested with *Diphyllobothrium latum,* probably only in 0.1 to 0.5 per cent of infestations. This anemia occurs almost exclusively in Finland (at least 70 per cent of the recorded cases), where the population has a predisposition to pernicious anemia and where, according to Birkeland, the tapeworm is probably a precipitating factor and not fundamental cause of the primary anemia, since many Finns who are cured

of bothriocephalus anemia later develop pernicious anemia. He, therefore, concluded that fish tapeworm anemia was not a clinical entity but an abortive form of true primary pernicious anemia. As we shall see later, however, this is probably not true.

As a rule, fish tapeworm anemia is indistinguishable from cryptogenic pernicious anemia clinically, hematologically and pathologically. Stomatitis and glossitis occur. Achlorhydria occurs in about 80 per cent of cases but hydrochloric acid may return to the gastric juice when the worm is expelled[21]. Achlorhydria is no more common in non-anemic cases than in the general population. Bothriocephalus anemia is commonest in the third and fourth decades, a younger age group than in Addisonian anemia, but is very uncommon in childhood. The blood and bone marrow appearances are identical in both conditions. The mortality was 100 per cent in fish tapeworm anemia until it was recognized as such and treated specifically with anthelminthics, when it dropped to 12 per cent[23]. This is much better at present with the addition of liver extract and Vitamin B_{12} therapy.

The reason why fish tapeworm infestation causes a megaloblastic anemia has been an important question for some time. In 1948 von Bornsdorff[25] aspirated tapeworm eggs via tubes passed into the small intestine and he reported that the worm was in the jejunum in all patients with anemia, whereas it was in the ileum in patients without anemia. It seems likely therefore, that the worm interferes with the interaction of the intrinsic and extrinsic factors only if it is in the jejunum. An explanation is thereby provided for the fact that not all tapeworm carriers have anemia. Reticulocytosis and red cell response follow removal of the worm, thus confirming that the presence of *Diphyllobothrium latum* causes the anemia. Still unexplained is the reason why so many patients cured of bothriocephalus anemia develop true pernicious anemia in later life.

Harland et al[21] state "it is emphasized that a diagnosis of idiopathic pernicious anemia should not be made under the age of 30 years without considering all the possible etiologic factors".

Until Bjorkenheim[26] recently made a study of the subject it was not confirmed that spinal cord degeneration could complicate this anemia. He found about 80 per cent of 75 patients with bothriocephalus anemia to have subacute combined degeneration of the spinal cord and peripheral nerve degeneration, which was not as severe as in pernicious anemia because the patients were of a younger age group and the duration was less. According to him expulsion of the worm resulted in extensive, frequently even complete, remission of the neurological manifestations. Additional treatment with liver extract or Vitamin B_{12} did not yield better results than a worm cure alone.

The problem of bothriocephalus anemia has not been encountered frequently in this country. Birkeland[23] in his extensive monograph on this subject states

"most cases of bothriocephalus anemia reported on this continent have been more or less sporadic and casual and have not contributed much to the discussion of this type of anemia". Most cases mentioned in the American literature are not well documented and often one cannot tell whether the anemia was primary or secondary in type. The impression from the literature is that there have not been many authenticated cases of tapeworm anemia in this country. Of 25 cases of infestation reported from the Mayo Clinic[4] in 1933, none had anemia. Barron[6] had one case of megaloblastic anemia in a man born in Finland. Hunnicutt[27] reported a Finnish-born male with a true primary tapeworm anemia, associated with achlorhydria, which responded to liver therapy with reticulocytosis and red cell response, although he still harbored the worm. Isaacs et al[28] had another typical case in a female born in Lithuania but in the United States

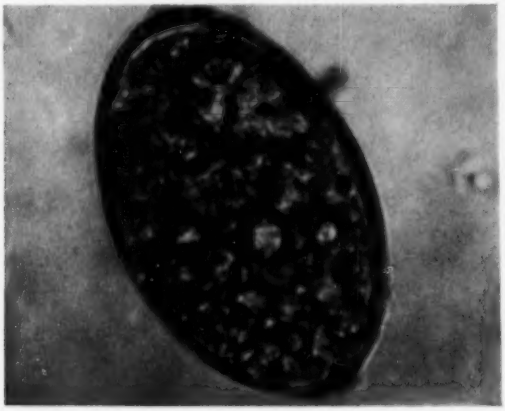

Fig. 4—*Diphyllobothrium latum* ovum in concentration of fresh stool specimen. Oil immersion, x 1,000.

43 years, who had achlorhydria and who responded to liver therapy. Of our 13 cases, 11 were studied hematologically and none showed any type of anemia and none revealed eosinophilia. An eosinophilia apparently is occasionally found but is inconstant[21]. Plotz[7] reported eosinophilia of 6 to 8 per cent in five of 11 cases. The consensus is that eosinophilia is not present as a rule.

Hernberg[29] was the first to visualize fish tapeworm radiographically. By means of a barium contrast meal in three previously known cases he visualized parts of the worm, one in the colon and, in two cases, in the ileum.

Diagnosis

The diagnosis of diphyllobothriasis should not be difficult. There are three principle questions to be considered: (1) Does the patient harbor a worm? (2) What kind of a worm? (3) Is the infestation cleared up after treatment?

Usually the patient comes to the office with the story that she has passed a worm. If she brings the specimen along it is easy to see whether it is a mucus cast, a round worm or a tapeworm. In some cases of mucous colitis with constipation, mucus casts, reaching occasionally a length of two or three feet, are passed. They are often mistaken for worms but on examination it can be demonstrated that they consist of longitudinal intertwining threads which can easily be worked apart with wooden applicator sticks. A small piece under the microscope shows the irregular formation and the many mucus cells they contain. Worms are fleshy and firm and can only be divided by cutting with a knife or scissors. Round worms are, when passed, complete, round and usually alive. Tapeworms are definitely flat and regularly segmented and the proglottids become larger the farther their distance from the head (Fig. 2).

In case no worm is brought to the laboratory, a purged stool specimen is necessary to confirm the infestation by demonstrating the ova. In contradistinction to ascarsis (roundworms), *taenia saginata* and *taenia solium* (beef and pork tapeworms), where often many stool specimens have to be examined before ova are found, eggs of fish tapeworms are usually plentiful and found in every specimen. They are broadly oval, 70 x 45 microns, light yellow, packed with steel-gray granules and moderately thick-shelled. They are operculated, and in freshly passed stools the operculum can only be seen as a slight indentation in the shell (Figs. 3 and 4). In older preparations the operculum opens sometimes, and can be easily distinguished.

When the patient brings a tapeworm to the laboratory, it has to be differentiated from the meat tapeworms, *taenia solium* and *taenia saginata*. As a rule the proglottids of *Diphyllobothrium latum* are much broader than they are long. Only the oldest proglottids, farthest from the head, are approximately square. The *taenia saginata* and *taenia solium* proglottids are either square or their length is greater than their width.

Another distinguishing feature is the fact that in the meat tapeworms often a single, motile proglottid is passed while in diphyllobothriasis the proglottids usually adhere firmly to each other and only longer pieces are evacuated in the feces. In every case it is best to confirm the diagnosis by identifying the eggs in the stool. Clearing the segments in glycerin and checking on the characteristic genital pores, uteri, etc., solves the problem, too, but it is time consuming and involves much more work. In the 13 cases in our laboratory, 12 were found because the patients passed the segments and one through a routine stool examination which disclosed the eggs.

In order to find out whether the treatment is successful, several stool examinations have to be done. It is of doubtful value to search for the characteristic head of the worm, as the patient can harbor several worms. The head is small, very soft, easily crushed and usually gets lost in the large amount of purged stool after treatment. We found it much more reliable to check three stools

at intervals of two to three days beginning five days after treatment. If no eggs are found in the last two specimens, the stools are rechecked about three months later. If one or more viable heads had remained in the intestinal tract they would have developed into mature worms and eggs would be found again. If this is not the case the infestation can be considered cleared.

Treatment

In the presence of anemia, liver extract or Vitamin B_{12} should be given and an adequate response obtained before treatment with an anthelminthic is considered.

In the past, oleoresin of aspidium, carbon tetrachloride and hexylresorcinol have been used, but they are toxic at times and must be used cautiously. The toxic effects of aspidium are usually due to individual idiosyncrasy and can include nausea, vomiting, gastroenteritis with sanguinous diarrhea, liver atrophy, convulsions, cardiac collapse and at times death. Therefore, it should not be used in patients with gastrointestinal, renal, liver or cardiac disease. It is contraindicated during pregnancy. Carbon tetrachloride is dangerous to use and must be employed with caution. It can cause hepatitis, coma and death. It is especially contraindicated in small bowel ulceration, nephritis, pregnancy, cases with fever and with serum calcium deficiency[5]. Hexylresorcinol is probably the least toxic of the three but its effectiveness has not been completely established for this infestation. Morales and Stevenson[30] reported that treatment with hexylresorcinol in the usual crystoid form is not satisfactory or effective but they administered to 28 patients with *taenia saginata* infestation an emulsion of crystalline hexylresorcinol directly into the duodenum through a duodenal tube with good results and no toxic effects. There was no need for a purgative or for dietary restriction.

Recently quinacrine (atabrine) and the related acridine derivative "Acrinal"[31] have been used in fish tapeworm infestation with success. They are especially good because of the low toxicity in the doses required, although certain possible side-effects must be kept in mind. These dyes have become the treatment of choice, after proving highly effective for beef tapeworm infestation. Morales[32] recently outlined the program of therapy in reporting the treatment of 24 patients with *taenia saginata* infestation. He gives 30 grams of magnesium sulfate at 3 P.M. the day before treatment. On the morning of treatment, no food is given, then the patient takes 6 to 8 atabrine tablets (0.1 gm.) for a total of 0.6 to 0.8 gm. depending on weight, 2 tablets every 5 minutes with a glass of water to which 6 gm. of sodium bicarbonate has been added (to prevent nausea and vomiting). After this, water or fruit juices are allowed freely. Two or three hours later another dose of magnesium sulfate is given, although this may not be required since occasionally atabrine will give adequate purgation. The segments are expelled alive and stained yellow. It is the opinion of the

author that the scolex is severely and sufficiently injured to release its hold on the mucosa of the intestine. Morales[32] had 96 per cent success. Sodeman[33] has treated 11 cases of *taenia saginata* infestation with atabrine along the same lines with excellent results and believes this is now the treatment of choice. Hoekenga[34] also used a dose of 0.8 gm. of atabrine to treat successfully 40 patients with *taenia saginata* and *Hymenolepis nana* infestations. "Acranil" may be given in a single dose or in two divided doses equivalent to 10 mg. per kilo of body weight. The drug is given orally in the morning, the patient not having eaten since the previous noon. A saline purge is given two hours after "Acranil"[31].

The most important toxic reaction in the use of atabrine for the treatment of tapeworm infestation is nausea and vomiting. In the Morales[32] series, six patients felt nauseated and vomited yellowish gastric contents within an hour after the last atabrine tablet had been given. Twenty per cent of Hoekenga's[34] patients had nausea and vomiting after atabrine ingestion and the intravenous administration of pyridoxine hydrochloride did not abort these symptoms. The above authors agree, however, that the nausea and vomiting produced by this drug is not very bothersome.

Of the more serious toxic manifestations encountered, Morales[32] had a patient who became listless, stuporous, incoherent and somewhat disoriented as to time and place after 0.8 gm. of atabrine. In 12 hours she had recovered from her psychiatric episode and a follow-up ten months later revealed her to be in good health. In Hoekenga's[34] series six people complained of transient dizziness and one six-year old boy developed a yellowish discoloration of the skin which lasted four days. One patient had a moderate neurological reaction for four days (sleeplessness, loss of balance, confusion and a positive Romberg test) but all symptoms cleared.

Morales[32] states "atabrine hydrochloride appears to exert certain toxic effects but not to any serious nature". A review of the literature indicates, however, that there are other toxic effects besides those mentioned above. Dockeray[35] states that serious toxicity is rare, but in therapeutic doses atabrine may cause vomiting, diarrhea, toxic psychosis, and tachycardia. He mentions a man who took 6 gm. in an attempt at suicide but with no sequelae, and another man who took 25 gm. in one dose and went into shock but recovered. He reports two cases of sick African infants who were accidentally given overdoses parenterally and died, both of heart failure. Atabrine should be given carefully to children.

Cameron[36] reports a case of acute psychosis after ingestion of 12 gm. of atabrine, which cleared in 48 hours without any residue. There are other toxic reactions which apparently occur only after prolonged administration, with the duration of therapy the determining factor. Thus atabrine in prolonged use may be a bone marrow depressant with hypoplastic anemias being reported[37], as well as causing toxic hepatitis with death[38], atypical lichen planus[39], aplastic anemia[40], atabrine dermatitis[41] with cutaneous atrophy, alopecia and anhydrosis

persisting for many years, exfoliative dermatitis, liver necrosis, and lichenoid dermatitis[42]. It is important to stress, however, that these severe toxic effects appeared in only a few of the millions taking this drug and then only after prolonged use, which is not necessary when used as an anthelminthic.

Six of our cases were treated successfully with 1 gm. of atabrine as suggested by Cecil and Loeb[43] and there were no toxic reactions, except for nausea and vomiting in one patient. Of these six patients, our five most recent cases were readily administered the atabrine via the Matzner polyethylene duodenal tube[44] to obviate any possible nausea and vomiting, which has been shown to be the most important side-effect of atabrine therapy; in spite of this one patient exhibited nausea and vomiting. The atabrine solution is prepared by pulverizing and dissolving ten 0.1 gm. tablets. This solution is not administered until alkaline-bile stained material is aspirated from the duodenal tube. The duodenal tube is flushed a few minutes after the drug has been introduced and then quickly withdrawn. The remainder of the program is similar to that outlined by Morales[32].

Report of Cases

Thirteen cases of fish tapeworm infestation have been diagnosed by us since 1950 (Table I). All patients were Jewish females and all had tasted raw "gefülte" fish during its preparation; one student nurse, 19 years of age had tasted it as a child, when her grandmother was cooking.

The ages ranged from 19 to 73 years. Four were born in the United States and the others were born in Poland, Hungary or Russia, except for the student nurse who was born in South America and came to this country 12 years prior to passing the worm. One 23-year old woman had come to this country only three months prior to examination. The other seven patients, however, had been in this country at least 23 years, one 66-year old female had come to this country at two years of age and a 42-year old patient at 3 years of age. One 73-year old patient had been in this country about 49 years. It is therefore probable that all of our patients, except the recent arrival from Europe, were infected in this country.

Twelve out of 13 patients came under medical attention because they had passed segments of the worm; nine of them brought the segments with them for diagnosis. The others gave a history of passing a worm and the diagnosis was made by finding the ova of *Diphyllobothrium latum* in the stools. Only six had gastrointestinal symptoms, consisting of abdominal pains, but one was the student nurse who also had amebiasis, another had a duodenal ulcer, while another had a poorly functioning gallbladder and two duodenal diverticula. One patient had anorexia, weakness and weight loss, but it was the opinion of the examining physician that her symptoms were psychogenic in origin and not related to her infestation. This patient did not pass segments but the ova were

TABLE I

	Sex	Age	Place of birth	Time in U.S.	G.I. complaints	Anemia	Eosinophilia	Associated G.I. disease	Treatment
1. S.B.	F	63	Poland	23 yrs.	#	0	0	G.B. disease 2 Duod. divert.	oleoresin of aspid.
2. B.B.	F	19	South America	12 yrs.	#	0	0	amebiasis	oleoresin of aspid.
3. I.F.	F	73	Poland	24 yrs.	0	0	0	0	none
4. B.L.	F	42	Russia	39 yrs.	0	0	0	0	oleoresin of aspid.
5. S.H.	F	50	Russia	36 yrs.	0	?	?	0	atabrine
6. P.C.	F	33	U. S.		0	?	?	0	hexylresorc.
7. B.P.	F	66	Russia	64 yrs.	0	0	0	0	hexylresorc.
8. M.R.	F	23	Poland	3 mos.	0	0	0	0	none
9. F.B.	F	45	U. S.		0	0	0	0	atabrine duod. intub.
10. J.S.	F	52	U. S.		#	0	0	0	atabrine duod. intub.
11. S.K.	F	50	U. S.		#	0	0	0	atabrine duod. intub.
12. H.G.	F	49	Hungary	30 yrs.	#	0	0	duodenal ulcer	atabrine duod. intub.
13. F.G.	F	62	Russia	42 yrs.	#	0	0	0	atabrine duod. intub.

found during a routine stool examination and she never received anthelminthic therapy. One patient was in the hospital for surgery of a benign breast tumor when she passed the worm preoperatively. No other complaints were noted by our patients.

In 11 of our 13 cases complete blood counts were done and there was no evidence of anemia or eosinophilia.

Six patients were treated with single courses of atabrine as described above. Repeat stool examinations were negative for proglottids or ova of *Diphyllobothrium latum*.

Prevention

All fresh water fish should be thoroughly cooked before ingestion. Heating at 65°C for five minutes kills the larvae of *Diphyllobothrium latum* and in ordinary cooking fish is subjected to 100°C or more for at least ten minutes. Cooks should be warned not to taste raw or half-cooked fish or its soup in connection with flavoring, even if it is not swallowed. Children should not be permitted access to fish markets or to kitchens when fresh water fish is being prepared because of the possibility that they may consume some uncooked.

Adequate freezing methods should be instituted in commercial houses that pack susceptible fish obtained from lakes in the United States and Canada known to be infested. Kajava[45] and Essex and Magath[46] have shown that submitting infested fish to a freezing temperature of 14°F (10°C) for from 28 to 48 hours kills the larvae of *Diphyllobothrium latum*. Apparently, smoked and canned fish prepared by commercial methods are safe as far as infestation is concerned[1].

Sewage in endemic areas should be treated with formaldehyde or chlorine before being discharged into lakes or streams. Public health inspectors should check samples of raw fish in markets just as is done with milk. Further surveys of lakes and streams should be made in order to discover the extent of infestation. As another protective measure to reduce contamination of waterways, dogs and cats should not be fed fresh water fish.

Summary

1. Thirteen cases, all in Jewish women, with fish tapeworm infestation are reported, all due to tasting inadequately cooked "gefülte" fish.

2. The life-cycle of *Diphyllobothrium latum* and the pathogenesis of fish tapeworm infestation are discussed. The lakes of Northern Michigan, Northern Minnesota and lower central Canada are implicated as the sources of infested fish, especially the fish sold along the eastern seaboard.

3. Most cases of fish tapeworm infestation in this country are asymptomatic and come to attention only upon passage of the worm. Fish tapeworm anemia is rare in this country but not infrequent in Finland. Current ideas on the cause of this anemia, which resembles pernicious anemia, are discussed.

4. The treatment of choice at present is atabrine, given orally in divided doses or through the duodenal tube for a total of 0.6 to 1.0 gm. The only important side-effect is nausea and vomiting and this may be minimized by duodenal administration, which is readily performed with the Matzner polyethylene duodenal tube. Other possible side-effects are mentioned. The serious side-effects of atabrine only follow prolonged therapy, which is not required in the treatment of this disorder.

5. Methods of prevention are mentioned, the most important being the adequate cooking of fresh-water fish before tasting, and the commercial freezing of susceptible fish from infested lakes.

6. Follow-up of three of our cases after duodenal instillation of atabrine indicated the absence of ova after several months.

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COMPREHENSIVE TESTING OF GASTRIC SECRETORY FUNCTION*

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Testing the secretory function of the stomach for diagnostic purposes has shown a progressive trend toward oblivion during recent years. This is apparently due to the limited inferences obtainable from the study of gastric acidity after histamine or various test meals, the diagnostic significance of which is much less than that of the good history and x-ray examination.

In evaluating the reasons for the diagnostic failure of the gastric analysis it appears astonishing that the blame was placed on the whole procedure and not upon the usual restriction of secretory studies to the determination of one

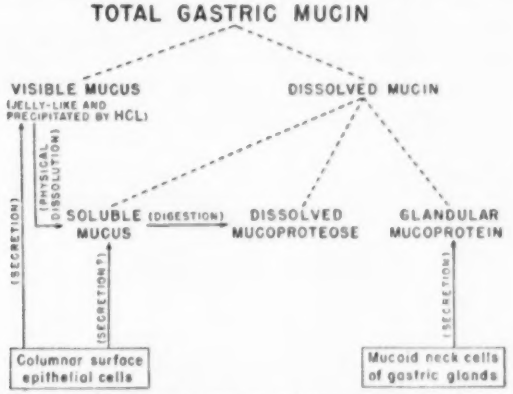

Fig. 1—Classification and derivation of mucus substances of the human stomach. (Glass: Gastroenterology, **23**:220, 641, 1953; Mersheimer, Glass, Speer, Winfield and Boyd: Ann. Surg., **136**:668, 1952).

secretory product, i.e. the acid. Since nowhere in modern medicine does the functional study of an organ become limited to testing of only one product of its metabolism by a single method, no wonder that the gastric analysis looks very inferior when compared to a comprehensive liver work-up or functional study of the kidneys.

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While in man the secretion of water and acid is influenced by both humoral and central nervous stimuli, the nonparietal components of gastric secretion, i.e. pepsin and mucus substances contained in the gastric juice are affected only a little by the humoral stimuli, but they are strongly dependent upon central vagal stimulation[1-4]. Therefore gastric physiologists for a long time have contended that testing of gastric secretion after only one humoral stimulus is not sufficient, and that at least two stimuli (one humoral such as histamine, and another vagal such as insulin) should be used for this purpose[3,4].

For several years we have attempted to extend the scope of gastric secretory studies by combining the histamine and insulin test and determining the

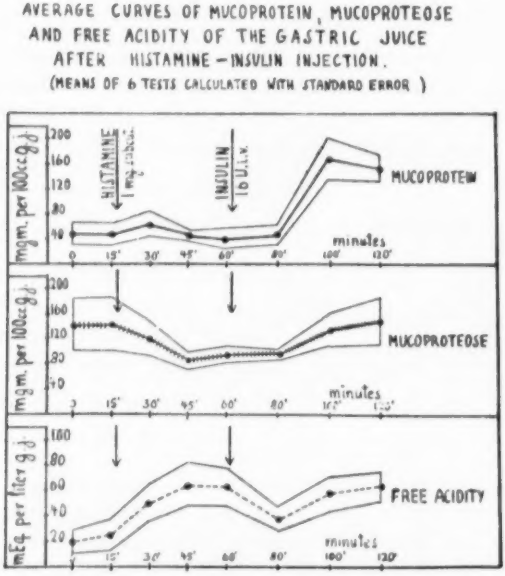

Fig. 2—Response of gastric acidity, glandular mucoprotein and surface epithelial mucoproteose to histamine and insulin stimulation. Each curve represents the mean value. (Glass: Rev. Gastroenterol. **16**:694, 1949).

acid and pepsin responses to both these stimuli. Moreover, we have also included the quantitation of the two main mucus fractions contained in the gastric juice into the gastric analysis[5-8].

Gastric mucin occurs in two forms. One is the visible mucus which is the secretory product of columnar cells of surface epithelial cells of the stomach; the other is dissolved mucin, which confers upon the gastric juice the feature of viscosity. The quantitation of the visible surface epithelial mucus cannot be included in the comprehensive study of gastric secretion, since owing to its

viscosity, most of it remains attached to the surface of gastric mucosa, and cannot be aspirated. We can measure, however, the rate of its secretion indirectly by determining the content of its degradation product in the gastric juice,

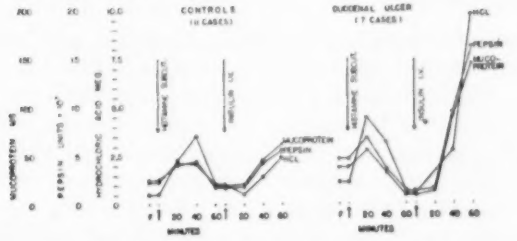

Fig. 3—Averaged recovery of hydrochloric acid, pepsin and glandular mucoprotein following histamine and insulin in 11 control subjects and 7 patients with duodenal ulcer. Each curve represents the mean value.

namely, the concentration and content of the dissolved mucoproteose[5] (See Fig. 1). This mucoproteose is one of the components of the heterogeneous complex of gastric dissolved mucin. Another component of the dissolved mucin is the dissolved mucoprotein[5] which we later called glandular mucoprotein[9] because

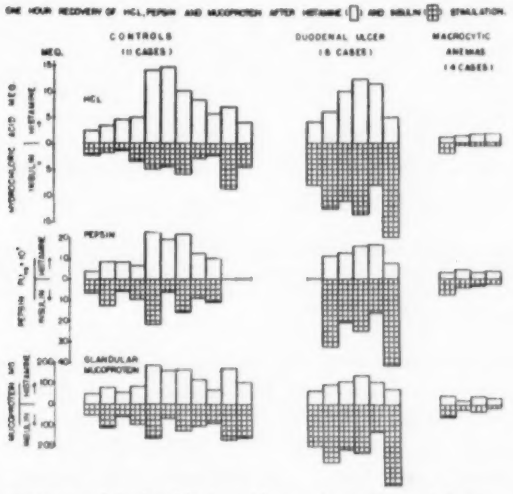

Fig. 4—One hour recovery of hydrochloric acid, pepsin and glandular mucoprotein following histamine and insulin stimulation in 11 control subjects, 6 patients with duodenal ulcer and 4 individuals with macrocytic anemia. Each column represents one case.

apparently it originates from the mucoid neck cells of the fundic glands in the fundus and body of the stomach (See Fig. 1). We recently found that it is either identical with Castle's intrinsic hematopoietic factor or that Castle's

intrinsic factor is contained in this mucus fraction[10,11]. The mucoprotein forms a complex with pepsin[4,12]. The secretory pattern of glandular mucoprotein often, but not necessarily always, parallels that of the pepsin[6]. Its secretion depends upon the integrity of gastric glands and it fails whenever these atrophy[13].

Our technic of comprehensive testing of gastric secretory function is as follows: Eight to twelve hours after the last meal the Levin tube is introduced into the stomach and the position of its tip in the lowermost part of the stomach is confirmed by fluoroscopy. Two fasting specimens at an interval of ten minutes are aspirated as completely as possible, and a dose of 0.1 c.c. of histamine phosphate per 10 kilogram of weight is injected intramuscularly. The specimens are collected at intervals of 20 minutes during the first hour after histamine injection. At the end of the first hour 16 units of regular insulin are

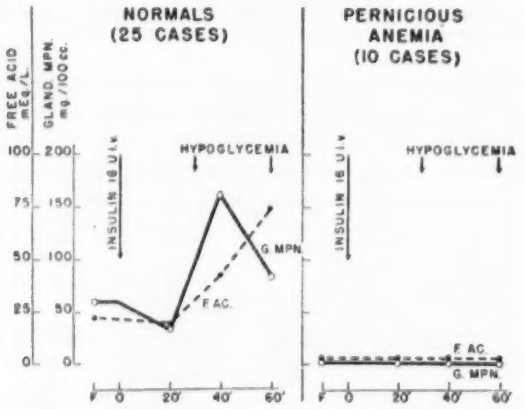

Fig. 5–Concentration of glandular mucoprotein and free acidity in the gastric juice of 25 normals and 10 patients with pernicious anemia under fasting conditions, and after intravenous administration of insulin. Each curve represents the mean value. (Glass: Gastroenterology, **23**:652, 1953).

injected intravenously and three specimens are withdrawn again every 20 minutes during the subsequent hour. In each of the specimens we determine: a) volume; b) pH, electrometrically; c) free and total acidity, by usual titration method; d) pepsin, by Glass-Pugh-Wolf modification of the Anson-Mirsky hemoglobin method[14], and finally, e) glandular mucoprotein and f) gastric mucoproteose by Glass and Boyd tyrosine colorimetric methods[5]. From the concentration of each of these components measured and the volume of each specimen, the total content of the particular component in each recovered sample is determined.

The purpose of the present communication is to demonstrate the usefulness of such a comprehensive study of gastric secretion. This paper is based on recent

data obtained on 55 individuals with various gastric pathology, as well as on our earlier studies comprising over 200 insulin and histamine tests[5-9,16,17,19-22].

Figure 2 shows the average acid, mucoprotein and mucoproteose response to the combined stimulation of gastric secretion with histamine and insulin. The

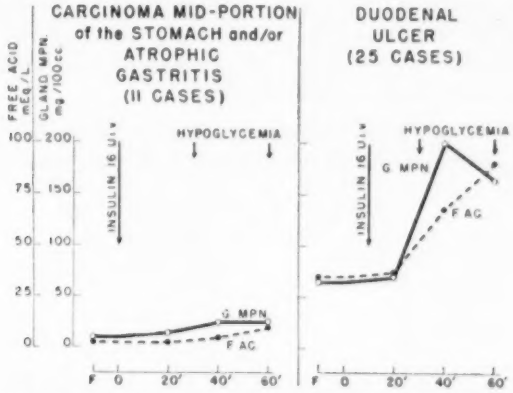

Fig. 6—Concentration of glandular mucoprotein and free acidity in the gastric juice of 11 patients with atrophic gastritis alone or associated with the carcinoma of the mid-portion of the stomach and 25 individuals with duodenal ulcer under fasting conditions and after intravenous administration of 16 units insulin. Each curve represents the mean value. (Glass: Gastroenterology, **23**:650, 1953; Mersheimer, Glass, Speer, Winfield and Boyd: Ann. Surg., **136**:668, 1952).

curves were obtained as averages from data collected in six normals and calculated and drawn with standard error. Two peaks of acidity will be noted,

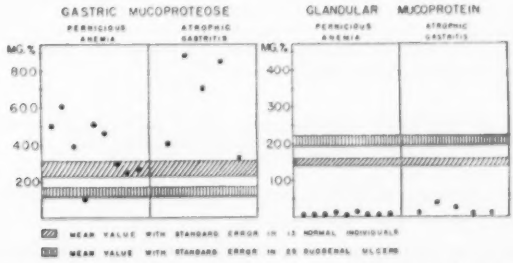

Fig. 7—Highest concentration of mucus substances in gastric juice after intravenous administration of insulin in 9 cases of pernicious anemia and 5 cases of atrophic gastritis. Each point represents one case. (Glass: Gastroenterology, **23**:646, 1953 and J. Nat. Cancer Inst., **13**:1016, 1953).

the first occurs within the first one-half hour after histamine, the second, 40 to 60 minutes after insulin injection. The acid response starts concomitantly with insulin hypoglycemia, but comes to a peak about 20 minutes later,

concomitant with the feeling of excessive hunger. Mucoprotein concentration shows a very slight rise after histamine, and a large increment 40 minutes after intravenous administration of insulin, concomitant with the hypoglycemia. Pepsin secretion shows behavior exactly similar to mucoprotein. As to the mucoproteose concentration, this shows a marked decrease after histamine, which is due to dilution of this surface epithelial product by the flow of gastric juice stimulated by histamine. After insulin, as a result of the effect of vagal stimulation upon this product, the concentration of mucoproteose returns to initial values, or even shows a moderate rise.

We observed this normal secretory pattern in many normal subjects, especially in the younger or middle-aged group, and also in many patients with

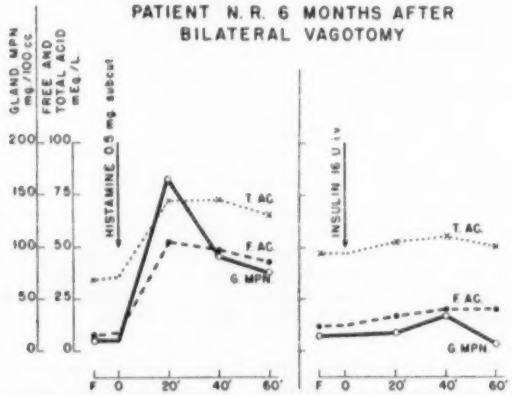

Fig. 8—Concentration of glandular mucoprotein and free acidity in the gastric juice of 25 normals and 5 patients following bilateral vagotomy under fasting conditions and after intravenous administration of insulin. Each curve represents the mean value. (Glass: Gastroenterology, **23**:651, 1953 and Mersheimer, Glass, Speer, Winfield and Boyd: Ann. Surg., **136**:668, 1952).

various diseases of the liver, gallbladder, small and large intestine, as well as functional disturbances of the stomach[7].

Patients with duodenal ulcer are notoriously hypersecretory individuals. Studied with our technic they demonstrate very uniform hypersecretory pattern, which is most evident during the insulin phase of our gastric secretory test.

The data in Figure 3 were collected in six patients with duodenal ulcer and eleven controls, who presented complaints of ulcer type without radiological evidence of peptic ulcer, but due to psychosomatic factors or gallbladder disease. The results obtained were averaged and drawn in the form of curves.

It is evident that the outputs of acid, pepsin and mucoprotein following histamine stimulation do not show any significant differences between both

groups of patients. During the vagal phase of the secretory test, however, i.e. after insulin, the outputs of all three glandular components, i.e. acid, pepsin and mucoprotein, in the group of patients with duodenal ulcer markedly exceed values observed in the control group.

Figure 4 illustrates these differences more exactly. The data were collected, as before, in patients with duodenal ulcer, in control patients with ulcer symptoms but without radiological evidence of ulcer, and finally in cases of macrocytic anemia associated with atrophic lesions in the stomach.

It will be noted that in the group of controls the hourly outputs of acid, pepsin and mucoprotein after histamine and insulin are similar. On the other hand the patients with duodenal ulcer differ from the controls in that the outputs

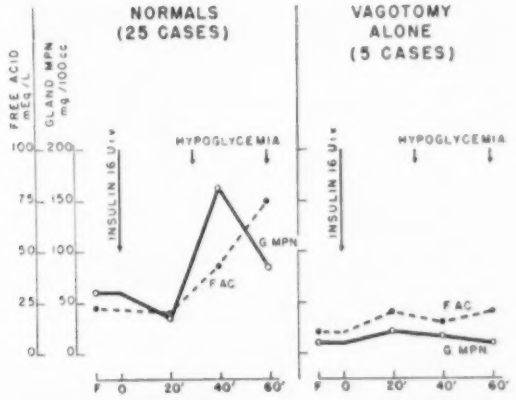

Fig. 9—Concentration of glandular mucoprotein and free acid in the gastric juice of a patient with bilateral vagotomy after intravenous administration of insulin and subcutaneous administration of histamine. (Mersheimer, Glass, Speer, Winfield and Boyd: Ann. Surg., **136**:668, 1952).

of acid, pepsin and mucoprotein during insulin phase markedly exceed those collected during the histamine phase of the test in the same individuals, and that they are statistically higher than corresponding recoveries in control patients.

Thus, patients with duodenal ulcer differ from other patients with similar gastric complaints but without ulcer, by a uniform hypersecretory response to vagal stimulation with insulin of hydrochloric acid[15], and also of the other glandular secretory products of the stomach, mucoprotein and pepsin[6,7]. It should be emphasized, however, that no significant differences between both groups are found during the histamine phase of the test, and that this hypersecretory pattern is found only in patients with duodenal, but not gastric ulcer, who behave in this respect similarly to the controls[22].

The pattern of gastric secretion in patients with gastric atrophy associated with macrocytic anemia, in sharp contrast to the hypersecretory pattern of patients with duodenal ulcer, shows extremely low outputs of acid, pepsin and mucoprotein both after histamine and insulin. The outputs represent only a small fraction of the recoveries observed in normals or patients with duodenal ulcer, and this is especially featured by patients with pernicious anemia, who uniformly have atrophy of cellular elements of gastric glands.

Figure 5 shows the negative response of acid and glandular mucoprotein to vagal stimulation with insulin in patients with pernicious anemia[9]. The flat curves of gastric secretion after insulin in patients with pernicious anemia are here contrasted with the usual rise in acidity and concentration of mucoprotein in normal controls.

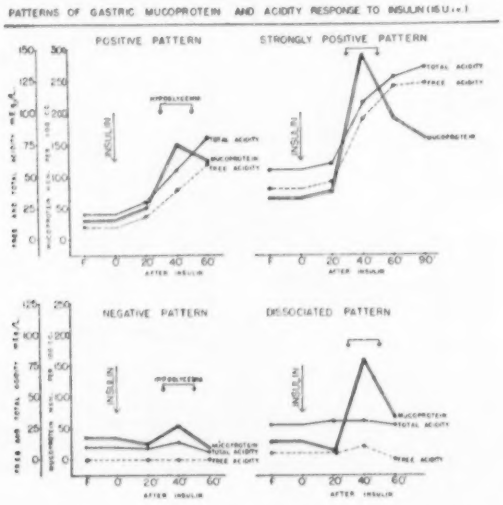

Fig. 10—Various patterns of response of acid and glandular mucoprotein to intravenous administration of insulin. (Glass and Boyd: Gastroenterology, **15**:446, 1950).

Figure 6 shows a similarly negative pattern of response of acid and mucoprotein to insulin in patients with carcinoma of the mid-portion of the stomach. The flat secretory curves of the gastric carcinoma are contrasted in this slide with the hypersecretory pattern of patients with duodenal ulcer. We found this flat response of acid and mucoprotein to insulin in 66 per cent of all 40 cases of gastric carcinoma which were studied[16], and nearly one-half of these cases were nonresectable.

In cases of atrophic gastritis diagnosed on gastroscopy and shown in Fig. 7, we also found the profound impairment of the mucoprotein secretion[17]. The

absence of mucoprotein after vagal stimulation in these instances indicates atrophy of the glandular elements of the stomach, and this finding was invariably confirmed, whenever the autopsy or biopsy of the stomach was performed. On the other hand the surface epithelial component of the gastric secretion, the mucoproteose, was present in all these atrophic stomachs in normal and often even higher than normal concentration. This was also true for gastric juices of patients with pernicious anemia and diffuse gastric carcinoma[16].

Another group of cases showing negative response of acid secretion to insulin are those after successfully performed bilateral vagotomy[18]. Figure 8 shows

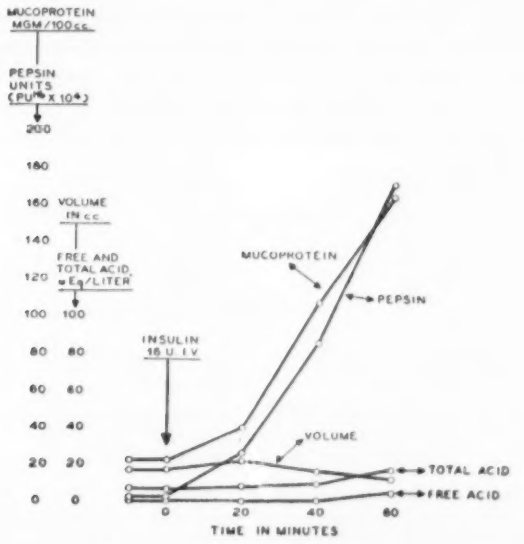

Fig. 11—Averaged response of gastric secretion to intravenous injection of insulin in 7 subjects following removal of the distal ⅔ to ¾ of the stomach for peptic ulcer. Each curve represents the mean value. (Glass and Wolf: Proc. Soc. Biol. & Med., **75**:536, 1950).

that the same holds also for the response of glandular mucoprotein to insulin[19]. The flat lines of mucoprotein and free acidity of the vagotomized stomach are contrasted in this slide with the usual response to insulin in normals. This negative response is the result of the severing of the efferent vagal pathways, which transmit to the stomach the stimulus, arising in vagal nuclei as result of insulin hypoglycemia.

The vagotomized stomach, on the other hand, might respond normally or almost normally to the humoral stimulation with histamine. Figure 9 shows data collected in one such patient studied six months after bilateral vagotomy, in

whom a negative response of acid and mucoprotein to insulin was obtained, but who showed a normal acid and mucoprotein response to histamine[20]. This dissociation between responses of vagotomized stomach to insulin and histamine demonstrate clearly that the vagotomized stomach is still able to produce acid and other glandular components in response to humoral stimuli.

The method of the comprehensive testing of gastric secretion allows one also to detect other types of dissociations which occur between secretory patterns of various products of the stomach. Some of these dissociations are very important from the diagnostic point of view.

Figure 10 explains the nature of one of these dissociated secretory patterns. The first pattern in this slide is called "positive pattern". This is characterized by the rise in acidity and mucoprotein secretion concomitant with the insulin

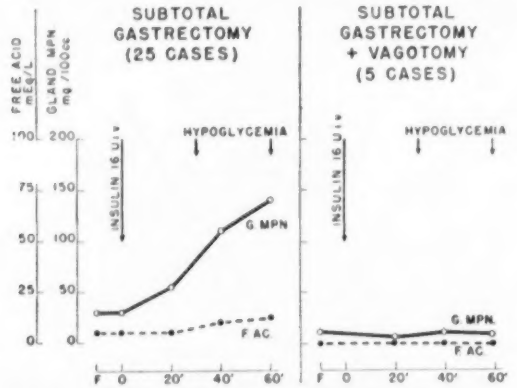

Fig. 12—Concentration of glandular mucoprotein and free acid in the gastric juice of 25 patients following subtotal gastrectomy and 5 patients following subtotal gastrectomy combined with bilateral vagotomy. Each curve represents the mean value. (Mersheimer, Glass, Speer, Winfield and Boyd: Ann. Surg., **136**:668, 1952).

hypoglycemia and it represents the normal response of gastric secretion to insulin. The second, called here "strongly positive" pattern, is characteristically observed in patients with duodenal ulcer. The third type, called here "negative pattern", represents responses observed in patients with pernicious anemia, atrophic gastritis, diffuse gastric carcinoma, or after bilateral vagotomy. It is characterized by complete or almost complete abolishment of acid, pepsin and mucoprotein response to insulin, and is due either to the atrophy of fundal glands, or to severing of vagi nerves to the stomach. The fourth secretory type, called here "dissociated pattern", is characterized by a very low or no response of acid to insulin, with a normally preserved response of other glandular components, i.e. pepsin and mucoprotein, to this stimulation. We observed this type of dissociation characteristically in patients following subtotal gastric resection[19],

in whom the pyloric part of the stomach has been removed, and in whom the remaining fundal and cardiac parts of gastric mucosa were well functioning and intact.

Figure 11 illustrates typical findings in these subtotally or partially gastrectomized cases[19] which consist in high concentration of pepsin and mucoprotein and low concentration of acid in 40 and 60-minute specimens after insulin injection.

It has been demonstrated in physiological work that this dissociated pattern may be explained as follows: The central vagal stimulation with insulin has a direct stimulative effect upon peptic cells of gastric glands producing pepsin, and mucoid cells of the neck, apparently secreting mucoprotein. This response is mediated directly by the discharge of the acetylcholin liberated at the vagal endings of the stomach. On the other hand, the stimulative effect of insulin upon the acid secretion, in addition to acetylcholine, requires another mediator, in form of a gastrin-like hormonal substance liberated in the antral area of the

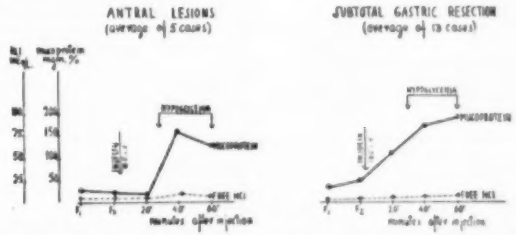

Fig. 13–Dissociated pattern of mucoprotein and free acid response to intravenous administration of insulin in 5 cases of inflammatory or infiltrative antral lesions and 13 patients with subtotal gastric resection for peptic ulcer. Each curve represents the mean value.

stomach under the influence of the discharged acetylcholine. Since this gastrin-like mediator is derived from the pyloric end of the stomach, the response of acid to insulin requires preservation not only of the vagal nerves to the stomach but also of the pyloric antrum in man[21].

This explains why, after partial gastrectomy with removal of the pyloric end of the stomach, the gastric secretory response shows preserved pepsin and mucoprotein secretion and reduced acid response to insulin[19], as shown in Figure 12. The curves shown here are the averages obtained from the study of 25 cases of subtotal gastric resection. It is worth noting that when subtotal gastrectomy is associated with bilateral vagotomy, the pattern of response changes into a negative one. This negative pattern we have described before as typical for the successful bilateral vagotomy[19,20].

We observed similar type of dissociation also in nonoperated patients in whom the distal area of the stomach suffered either from neoplastic infiltration

or extensive inflammatory or atrophic lesions[16]. This is shown in Figure 13, which illustrates dissociation between positive mucoprotein and negative acid response to insulin in 13 cases after subtotal gastric resection and in five non-operated cases with localized antral lesions, such as prepyloric benign ulcer, antral gastritis, or circumscribed neoplastic infiltration of the pyloric or prepyloric area.

The last group is most interesting in this connection and Table I shows data on mucoprotein secretion obtained on 40 patients with gastric carcinoma[16]. You will note that out of 21 cases of gastric carcinoma in whom no mucoprotein secretion could be detected after insulin, in 19 the lesion was diffuse and invading the entire body and fundus of the stomach of gastric carcinoma and

TABLE I

MUCOPROTEIN SECRETION AND LOCALIZATION OF THE GASTRIC CARCINOMA*

Glandular mucoprotein in gastric juice after histamine and insulin	Number of cases of gastric carcinoma studied	Localization of carcinoma in the stomach	
		Diffuse	Circumscribed to cardiac or prepyloric area
Absent	21	19	2
Greatly reduced	6	4	2
Normally preserved	13	2	11
Total	40	25	15

*The table is based on data published by Wolf, S., Glass, G. B. J., McNeer, G. and Bowden, L.: *J. Nat. Cancer Inst.* **13**:1025, 1953.

that the same was true in four out of six cases in whom mucoprotein concentration was greatly reduced. On the other hand out of 13 cases of gastric carcinoma in whom mucoprotein secretion after insulin was normally preserved, 11 had a circumscribed lesion localized to the cardiac or pyloric area of the stomach, and not invading the sites of secretion of mucoprotein, i.e. fundus and body of the stomach[16].

Therefore, one can conclude that when glandular mucoprotein is present in the gastric juice of patients with gastric carcinoma in normal amounts, following histamine and/or insulin stimulation, there is a chance 85:100 that the lesion is circumscribed to the cardiac or pyloric end of the stomach. When glandular mucoprotein, however, is absent entirely from the gastric juice of patients with gastric carcinoma, following histamine and/or insulin stimulation, there is a

chance 85:100 that the lesion infiltrates diffusely into the mucosa of the fundus and body of the stomach.

We have observed another type of dissociation in patients with localized lesion of the prepyloric or pyloric area of the stomach, which consists in that the acid response to histamine is much higher in the same individual than to insulin. Out of ten cases listed in Table II, with the exception of the last two showing similar responses to both stimuli, in eight the acidity response to histamine was much higher than to insulin, which often was entirely absent.

TABLE II

HIGHEST FREE ACIDITY AFTER HISTAMINE AND INSULIN IN 7 CASES OF CIRCUMSCRIBED GASTRIC NEOPLASM LOCALIZED AT THE PYLORIC END OF THE STOMACH*

	Histamine (0.1 mg./10 kg. subcut.)	Insulin (16 units i.v.)
1. Adeno-ca. gelat. gr. 4 (lower half lesser curv.).	107	0
2. Adeno-ca. gelat. inop. (distal ⅓).	45	0
3. Lymphosarcoma (distal ⅔ ant. & post. wall).	40	0
4. Adeno-ca. gr. 2 (antrum).	67	44
5. Adeno-ca. gelat. (prepyloric).	86	43
6. Adeno-ca. gr. 2 (pyloric end of lesser curv.).	70	27
7. Reticulo-cell sarcoma (lower part of the antrum).	80	43
8. Adeno-ca. gr. 3 (lower part of greater curvature and body).	45	0
9. Adeno-ca. gr. 2 (prepyloric).	0	0
10. Adeno-ca. gr. 2 (pyloric).	100	96

*The table is based on data published by Wolf, S., Glass, G. B. J., McNeer, G. and Bowden, L.: *J. Nat. Cancer Inst.* **13**:1025, 1953.

The explanation is that enough of parietal cells are preserved above the infiltrated prepyloric or pyloric lesions so as to respond directly to histamine stimulation. The response of parietal cells to insulin, however, is decreased or abolished because the distal area of the stomach which produces the mediating gastrin-like hormone necessary for insulin response of acid has suffered from neoplastic or inflammatory lesion[21].

Table III summarizes data on correlation of acidity response to histamine and insulin in the same patient in various diseases. In normals as well as in

patients with various secretory and motility disturbances of the stomach, the size of the acid response to histamine and insulin is approximately the same. The same is true for cases of diffuse involvement of mid-portion of the stomach or gastric atrophy in whom neither of these stimuli produces a significant amount of gastric acidity. On the other hand, in duodenal ulcers, the insulin produces much higher acidity values than histamine, and in the neoplasms of the distal end of the stomach, vice-versa the response of hydrochloric acid to histamine is significantly higher than to insulin.

In conclusion, it appears that the comprehensive testing of gastric secretory function after both humoral and vagal stimulation is of great importance for the evaluation of the functional status of the stomach, and the extent, nature and

TABLE III

RESPONSE OF GASTRIC ACIDITY TO HISTAMINE AND INSULIN IN VARIOUS DISEASES OF THE STOMACH

Gastric pathology	Number of cases	Highest free acidity in m. eq./liter (mean with standard error)	
		After histamine	After insulin
Functional secretory and motility disturbances, hypertrophic gastritis, postcholecystectomy syndrome.	11	75.0 ± 5.4	80.0 ± 7.2
Duodenal ulcer	6	83.0 ± 6.8	114.0 ± 6.2
Neoplastic involvement of the distal end of the stomach.	10	64.0 ± 10.2	26.0 ± 10.0
Neoplastic diffuse involvement of the mid-portion of the stomach	12	4.0 ± 3.7	4.0 ± 2.3
Gastric atrophy in pernicious anemia	10	0.0 ± 0.0	0.0 ± 0.0

localization of the underlying anatomical lesion. The inferences obtained from the secretory response have invariably proved correct on radiological or gastroscopic examination, as well as on biopsy or at autopsy.

Since the insulin test is completely harmless as indicated by our experience in over 250 insulin tests performed without a single untoward incident and since the whole procedure requires only about two hours, and the various measurements can be done in any clinical laboratory, the method is entirely suitable for routine clinical use, and even private practice. The comprehensive secretory test of gastric function in this form deserves an equal position to that of the tests of liver or kidney function, and ceases to be the poor relative of the rich family of functional tests used in internal medicine.

Discussion

Dr. Stanley Stark (Brooklyn, N. Y.):—This is a question for Dr. Glass: How long and how difficult is it to determine the contents of mucoprotein and mucoproteose in the gastric juice, and are these methods applicable to the average laboratory?

Dr. Glass:—Well, I can say that the determinations of mucoprotein and mucoproteose in the gastric juice are similar in scope to that of serum protein fractions by the tyrosine method of Greenberg. The time consumed for this test is approximately equivalent to that which has to be spent in the laboratory for the estimation of the albumin/globulin ratio by any of the available chemical (not electrophoretic) methods. The method of the determination of mucoprotein and mucoproteose fractions in the gastric juice is, therefore, perfectly applicable to any of the average clinical laboratories.

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A CONSIDERATION OF GASTRODUODENAL DISORDERS*

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The medical student who reads the various textbooks and journals assigned to him for the study of the gastroduodenal disorders understandably finds himself confused when he tries to apply his learning to the service of his patients on the ward. He finds himself hampered, mostly by the differing views of the various segments of the medical profession, even though the ultimate aim of all these segments may be the eventual best recuperation and maintenance of the patient. The advanced physician gains in his ability to serve his patients as his experience broadens and his interest and facilities enable him to apply his knowledge. The patient's welfare ultimately depends on whether all the forces of knowledge can be brought to bear to keep him in, or restore him to, a normal and healthy state, and enable him to live to his best advantage.

In the past 150 years, surgery, pathology, physiology, internal medicine, and other specialties too numerous to mention have all contributed to our knowledge of peptic ulcer. Some of the problems in deciding the management of the gastroduodenal cripple have arisen from conflicting programs of those interested in his care. As the specialties develop along lines of activity, instead of centering themselves about the patient and his total problems, it might be imagined that the various branches of medicine could lose sight of one another's aims, individual gains and progress. The physicians most likely involved with the gastroduodenal cripple and his problems are the family doctor, the internist, and the surgeon, but the radiologist, pathologist, and psychiatrist, the physiologist, biochemist, and welfare worker, the personnel manager and others are concerned with this disease.

Deaths due to peptic ulcer disease have shown an increase in this century, and certain studies indicate that roughly 7 per cent of the adult population can be shown to have duodenal ulcers demonstrable by x-ray, either active or in a "healed" state[1]. Three to 19 per cent of the autopsy material in various series show demonstrable active ulcers or ulcer scars[1]. It would seem that the chronicity and generally mild nature of the disease account for much delay in diagnosis and specific treatment of these illnesses. One can only guess at the true incidence of this disease in its various milder forms.

If a suitable standard program of patient observation could be developed, a basis could be provided that would allow true evaluation and comparison of the thoughts and experiences of the various sections of our present day medical organizations. In these days, when the patient is offered a raft of remedies, from

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cabbage juice[2] to surgery, there is obviously a need for some clarification and standardization of disease concept and treatment evaluation. If more than one method of treatment can be used satisfactorily for the disease, or one of its phases, then that fact ought to be recognized. If past, present, or future concepts lend harm to the status of the patient, then it follows that the earlier this is realized and the profession alerted to the situation, the sooner will the patients share in a beneficial effect. The author believes that all sections of medicine can contribute to the gastroduodenal cripple's welfare, but that a union of forces and interest is necessary to gain the best advantage. As in any social, political, or scientific gathering, there is a need for a common language so that the members can channel their thoughts, so they can understand and convey their impressions and contributions to one another. In order to facilitate discussions as to what constitutes the problems of the mid-twentieth century gastroduodenal cripple, a descriptive classification will be offered, which might serve to clarify the individual patient's problem in regard to his disease. The author feels that too much emphasis has been placed on the disease itself, without regard to what has happened to the patient generally, because of his disease. Since there is a great variety of subjective and objective findings related to these disorders, and since they are not always consistently correlated with the eventually recognized state of affairs of the patient, there is a great need for a practical approach to the various problems of the gastroduodenal cripple. Regardless of whether, for example, an ulcer niche of a certain size is demonstrable, or a gastric analysis test registers a certain high, it is in the last analysis, most important to know, whether or not the patient is faring well nutritionally, socially, physically, as a result of the ability or lack of ability of his or her gastroduodenal segment to function in a physiologically satisfactory manner. Regardless of any hormone or other physiochemical relationship the gastroduodenal segment has to the body economy, there is one obvious function that rarely receives great discussion, perhaps because of its apparent acceptance without second thought, and that is its function as a portal of entry for food. Recent discoveries in the fields of endocrinology[3], nutrition[4], and body chemistry[5] and dependence on at least the ingestion of food with its essential elements or component compounds lends great weight to the need for maintaining this function of the gastroduodenal segment. Treating the ulcer to the detriment of the body would be comparable to a farmer not admitting tractors, men, and fertilizers for use on his fields because his gate to the farm creaked every time it was opened. If the ulcer or the treatment of the ulcer deprives the body of its necessary nutrient materials, then it might be wise to treat the body and not the ulcer.

What is Peptic Ulcer Disease?

The pure scientist may rebel at the thought of a treatment for a disease, without understanding the processes that lead to the disease. It would be welcome if this were possible, but in truth, we must be content often with restoring

a patient to useful function without always knowing exactly what has transpired. Our concepts of cancer, for example, may be wrong, but surgery and medicine have much to offer, including various hormonal influences, whose mechanism of action may be explained differently in any short time, but which represent our attempts to restore the cancer patient to a state of well being. The plan of treatment for the gastroduodenal cripple ought not do less.

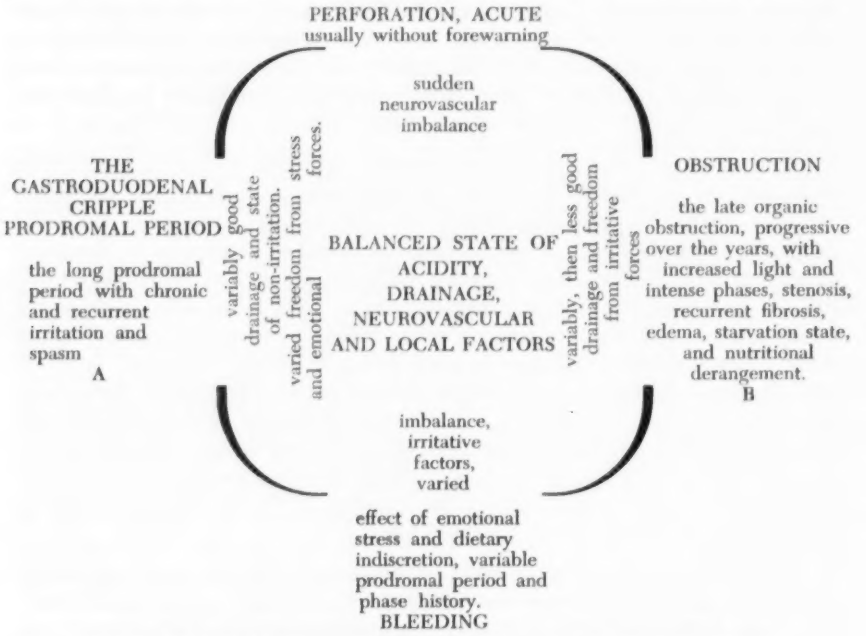
There have been several theories advanced as to the cause of gastroduodenal disease[6]. Plans of treatment have often been patterned after these well known concepts of hyperacidity, vagus nerve irritability, mucus deficiency[22] and perhaps others I may have failed to mention. Lesser hailed concepts, but with perhaps some important degree of weight for concern, are such considerations as local vascular deficiency, bacterial inflammation, anatomic irregularity, psychic stimulation, stress factors[21], and nutritional deficiency[23].

In such a situation, where innumerable possibilities exist, would it not seem wiser to consider all the factors as they seem to apply in any individual case? In any given population group, the patients who have been given the diagnostic term of "Peptic Ulcer Disease" might more accurately be described as to their complications and be better evaluated for it. The treatment or outcome of untreated bleeding or perforated peptic ulcer is far different from the minor digestive disturbances that are often called "ulcer" and dismissed with a minor effort at treatment. A complication that is not often recognized in peptic ulcer disease, because of its very chronicity and minor symptomatic disturbance is starvation. There is evidence that vitamin deficiency[7] and nutritional disturbance[8] can follow prolonged treatment of peptic ulcer disease with diet restriction. If hormonal effects can follow starvation[3], then is there not a folly to any method of treatment which ignores the function of the stomach in conveying foodstuffs to the hollow of the intestinal tract?

While not completely accepted by all physicians[9], there is much support for the concept of transition of an ulcer of the gastric wall into a cancerous lesion[10]. This factor has been a basis of concern in attempting to map a selected program of care for these patients, based on statistics[11]. It must be realized, however, that there is probably an individual factor in the patient's peculiar malignancy that has to do with the rate of growth. Nevertheless, until these mysteries are solved, we may have to rely on statistics to guide us in our over all judgment. There is no doubt that genetic forces may have some as yet undisclosed force that influences the individual case[12]. Some would attach significance to dietary habit, on a national basis, but would not national groups in cohabitation allow genetic influences to be equally effective, or at least in some manner, selective?

It would seem most practical, then, to consider the peptic ulcer patient as one who suffers from a disorder of the gastroduodenal segment, usually exhibiting irritative or spastic phenomena that can result in ulceration, recurrent ulceration, healing, erosion of a vessel with resultant bleeding, necrosis of a wall with

resultant perforation or penetration, or reparative forces with fibrosis, and in some cases narrowing with obstruction. The degree to which his disease carries him toward illness or death can vary with each case and at surprisingly different rates. The effectiveness of treatment, whether medical or surgical, cannot be predicted in the individual case, and it is this fact which would seem to suggest that, though generalizations can be made, and perhaps we can eventually agree on the best program for certain complications (perforations[13], bleeding in the aged[14]), the over all treatment of any patient with peptic ulcer disease, must be


PERFORATION, ACUTE
usually without forewarning
sudden neurovascular imbalance
THE GASTRODUODENAL CRIPPLE
PRODROMAL PERIOD
the long prodromal period with chronic and recurrent irritation and spasm
A
variably good drainage and state of non-irritation.
varied freedom from stress and emotional
BALANCED STATE OF ACIDITY, DRAINAGE, NEUROVASCULAR AND LOCAL FACTORS
variably, then less good drainage and freedom from irritative forces
OBSTRUCTION
the late organic obstruction, progressive over the years, with increased light and intense phases, stenosis, recurrent fibrosis, edema, starvation state, and nutritional derangement.
B
imbalance, irritative factors, varied
effect of emotional stress and dietary indiscretion, variable prodromal period and phase history.
BLEEDING

A and B probably represent portions of a vicious cycle, B conceivably the end result of earlier forces of irritation and spasm, and later edema, fibrosis, and stenosis.

Fig. 1

decided as to effectiveness in terms of whether it is restoring him to useful life and work, and a sense of well being, in a manner that does not harm his general bodily welfare. Figure 1 illustrates the generally considered forces at work.

Certain experiences and findings in a personal study of 100 cases classed as peptic ulcer disease will help clarify the features of the Descriptive Classification and help point out the need for a program of observation, classification, education (of patient, and physician), and a concept of "fitting the treatment to the patient", rather than fitting the patient to the treatment favored by any particular

person. I believe it may be a common fault of the average physician to assume that, because a well-known internist or surgeon illustrates a method of management or a surgical technic, that the internist or surgeon concerned treats all patients with peptic ulcer disease in the same manner. It is my own observation that the experienced internist, surgeon, or general practitioner actually does much selection in choice of treatment for his patient and that the disinterested

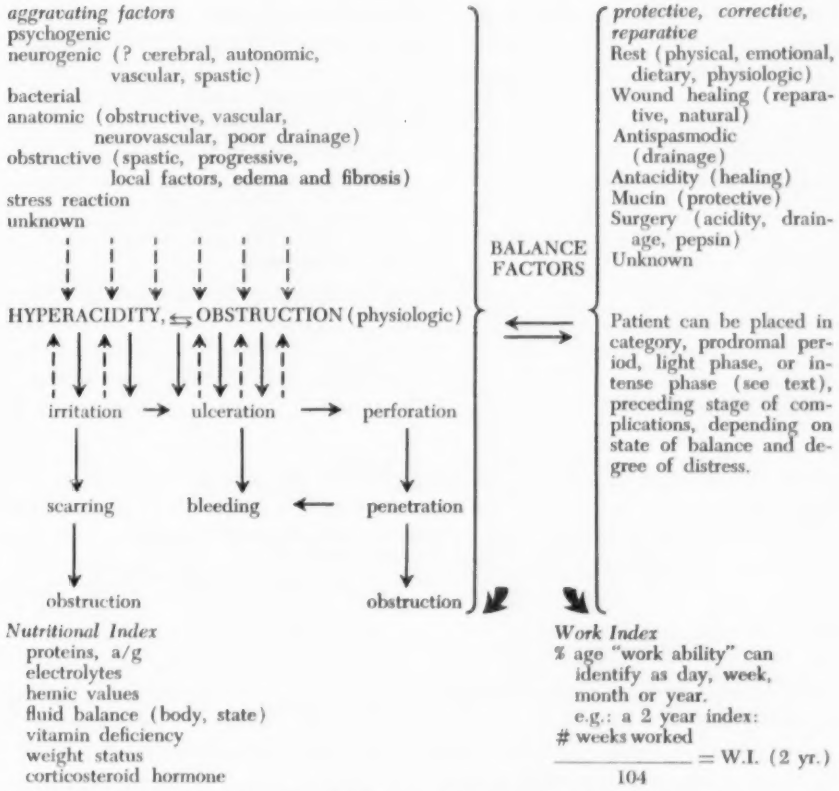


Fig. 2—Attempts to describe the factors at play in affecting and describing the life history of the gastroduodenal cripple patient, centering on hyperacidity and obstructive factors, but noting the eventual reliance on a nutritional and work index as clues to the true status of the patient in coping with his disease.

physician, regardless of how well trained he may be in other matters, may do harm by too insistent adherence to a proclaimed effective medical or surgical means of therapy.

The Descriptive Classification suggests that there is a varying degree of intensity, and rate of disturbance in any one individual case and the state of

effectiveness in counteracting the disease processes determines any need for change or further treatment. What is meant by effective treatment may vary from time to time and be dependent on future concepts and discoveries. Figure 2 further elaborates upon the gross terminology of the Descriptive Classification. A Progressive Evaluation Chart, for individual study of the patient as the years go by, combines the essential features of Figures 1 and 2.

PROGRESSIVE EVALUATION CHART—GASTRODUODENAL

Name age work MSWD Children

Section I in general, A Excellent
B Anaprogress (positive balance)
C Cataprogress (negative balance)
D Poor

Weight hold, or gain A B C D

Strength status A B C D

Work Index
A (100%)
B (80%)
C (60%)
D (under 60%)

Subjective comfort, distress
A (No distress, no medical care)
B (controlled with medicines and diet)
C (med, diet, and rest from work)
D (no relief)

Nutritional Index
A (good food intake, status A in above)
B (modified diet, status B in above categories)
C (limited diet, danger of food deficiency)
D (obvious food lack, abnormal chemistries, protein, others)

Frequency and duration, of medical care
A ill less than 6 total days a year
B 2 to 6 office visits a year, controlled
C 6 or more visits a year, office, uncontrolled
D Constant care, hospital type case

Section II
Need for further present studies, consultation, and differential diagnosis
Radiologic, gastric analysis, gastroscopy, stool studies,
diff diag: esoph varices, portal hypertension, pancreas, gallbladder, diaph hernia, other, psychiatric

Section III—Factors in Background
Adequacy of investigative studies
Excessive "D" values in section I ?
Duration—prodromal, light, intense phases
Complications: perforation, bleeding, obstruction, starvation, past surgery, failure of medical care
Aggravating factors (see Figs. 1 and 2)

Section IV Physician Evaluation of patient
Diagnosis confirmed?
Satisfactory conservative alternative medical adjustment for care?
Patient's opinion
Patient controlled?
Consultant in order?
internist, surgeon, gastroenterologist
psychiatrist, social worker, other
Status of work index and nutritional index

Plan of treatment and date of next evaluation_____

The Descriptive Classification:—The Prodromal Period, the light phase, the intense phase. The degree and type of disturbance (pathologic, physiologic, anatomic). The Work Index, The Nutritional Index. The Time Factor (recurrence, continual, duration).

This classification should allow the pinpointing of a patient in an aura of comprehension and allow the physician to better evaluate his patient and direct his care.

The Prodromal Period is described as that during which the gastroduodenal segment exhibits some degree of disturbed function, but this is mild enough to be compatible with normal living and normal activity. Many have a history of childhood allergy to spiced foods, nuts, or chocolate, or having been told that they had a tender or nervous stomach. The subject does not seek medical care in this period, but if a youth or child, his parents may note his delicate approach to food and take him to the doctor. Many adult patients noted a sudden weight gain when they discovered that ice cream, often eaten in huge quantities, gave them a unique sense of well being in the epigastric region. One patient was relieved of night epigastric pain following subtotal gastrectomy for sudden, massive, recent bleeding from a duodenal ulcer, and had not sought treatment for over 20 years, thinking that the night pain was common to all normal people. Some gave a peculiar history of "killing the pain" with whisky and noted that later effective medical or surgical treatment eliminated the need for, or desire for, alcohol. It was noted that several patients, while not seeking medical advice, learned to restrict themselves in activity, exertion, excitement, and diet, and essentially provided themselves with what is often the sole program of therapy of many physicians in the treatment of these disorders, especially in the light phase.

In the Light Phase, the characteristics of the disturbance become more recognizable and the patient becomes aware that he is not in a happy state. The symptom complex[15] is usually recognized by his attending physician and treatment instituted, sometimes without the benefit of attendant x-rays or laboratory studies, but usually followed by a period of relief.

In the Intense Phase, the patient begins to note a severe interference with his ability to carry out his duties and is unable to gain the nourishment he expects. His disturbance may be related to pain, emesis, weight loss, weakness, or bleeding. In this phase, the patient becomes concerned and seeks medical help in order to maintain or regain his ability to carry out his ordinary activities.

The Time Factor may become very important since the prolonged duration or recurrence of the disease state, begins to take its toll on the work efficiency and nutritional status of the individual.

A patient may enter the Intense Phase suddenly with the appearance of massive hemorrhage, perforation, or obstruction. Intensive medical or surgical help in these instances can often return the patient to a satisfactory state whereas those with a Light Phase may suffer for decades, with half-hearted attempts at therapy on the part of the patient and sometimes the physician. Any age and either sex can be affected.

Application of a *Work Index* and *Nutritional Index* can guide a physician in his choice of treatment for certain patients who might otherwise not be considered suitable candidates for a chosen method of treatment. Examples of this will be given later, in this discussion. These terms are essentially self explanatory. The Work Index is reflected in the ratio of actual against expected work-ability of an individual. The time factor indicates the period involved. The Nutritional Index is a more subtle definition and today might include protein studies, weight changes, body fluid state, hematologic values, vitamin levels, enzymatic factors and even other considerations as technical knowledge and laboratory facilities increase. What we know today is probably only a small part of the complex interrelationships of the living processes. Would nutritive deterioration affect adversely the living processes of tissue repair, immunologic and allergic phenomena that form a part of other disease problems? It would seem so.

Phenomena of Irritation and Spasm

It was interesting to note that several patients who eventually were accepted as bona fide peptic ulcer patients because of x-ray demonstrable duodenal or gastric ulcer had long before, and in some cases several times, demonstrated on x-ray studies, a spastic or an irritable duodenum. Mass studies in large groups of these light phase patients could help determine prognosis and whether intensive treatment would be warranted in attempting to reduce the over all death rate from peptic ulcer disease, as compared with occasional treatment in this group.

Whether spasm is a protective factor or an etiologic agent is difficult to determine. This can be said in view of a case, JM described as follows:

History:—18-year old soldier, admitted to the hospital with a 2-hour history of severe upper abdominal pain, sudden onset, no previous history of pain or nausea. Examination revealed a "Board Belly" type of abdomen. Flat plate of abdomen questionable for air under the diaphragm (RSU Film). Preoperative diagnosis: Perforated Peptic Ulcer.

Surgery report:—Exploratory negative except for a hard "broomstick-like segment" of the first part of the duodenum. No gross evidence of perforation or fluid escape. No free air in the peritoneal cavity.

It was decided to flow 2 per cent sterile procaine solution over the hardened segment. This 4 x 2 cm. segment, 2 minutes later became doughy and its pink color was interrupted in the anterior central portion by the appearance of a 1 cm. whitish disc area. The disc area a minute later became pink and thereupon a central punched out hole appeared, from which exuded a frothy, clear or slightly cloudy fluid which was assumed to be gastric juice. Closure in the usual manner was followed by an uneventful course.

Later history was offered by the patient that he had suffered a severe emotional trauma, for 48 hours prior to the perforation, related to receiving an unexpected letter from his fiancee, 3,000 miles distant, that she had eloped with another man. He had never had any gastrointestinal complaints or abdominal pain prior to the present illness.

As regards the above case, it would seem that application of the procaine allowed us to review in reverse the sequence of events that may have occurred in the development of the perforation. If this is so, then one might assume that after the initial psychic trauma had somehow affected the local tissues to set the stage, there developed: perforation (preceded by ulceration?), disc blanching, segment spasm and contraction. Is spasm a protective phenomenon? Is there a subtle difference between irritation and spasm?

Observations and Experiences

While reference has already been made to certain findings in the group of 100 cases studied, it would be well to summarize certain unusual tendencies in the histories of these patients. Since the search was limited to 100 cases, and not confined to any one characteristic, this is not offered for its statistical value. Nevertheless, certain findings indicate the need for careful evaluation of the gastroduodenal cripple, lest his treatment or lack of treatment bring him further harm. It is also suggested that lack of common action, concern, or contact harms the physician-patient relationship and thereby the patient. It is also probable that use of the Progressive Evaluation Chart in mass studies would aid in study ing the clinical development of these diseases and form a basis for their standardization in concept, care, and prognosis.

Fifty-eight per cent of the patients had an intense phase of 2 or more years' duration. Nine of these patients had a history of 2 or more years' strict limitation to a milk and water diet as a medical regime (physician approved). Three of the nine had limitation for most of 7, 4, and 4 years with supplemental vitamin therapy and in them, no x-ray studies had been considered necessary, "because the patient was all right, and didn't vomit or have pain if he stayed on his diet". Four obstructed and 2 carcinoma patients were in this group of nine.

Twenty-nine came to surgery and had an intense phase of 2 or more years' duration, and these were personally operated and followed. Twenty-nine more of the remaining 62 had an intense phase of 2 years' or more, but had not been considered as having received adequate medical trial, and had a work index of at least 90 per cent. The operated cases, on the other hand, had work indices of 60 per cent or less for at least 6 months, except the cases which showed perforation.

Of the 4 perforations, 3 were duodenal and had a work index of 100 per cent except for the day of perforation, and had no light or intense phase. Two of

these later could be evaluated as having had a prodromal period since infancy but none could be determined for the case already presented, JM. The fourth perforation was of the lesser curvature and this patient had an intense phase of 1 week, and a work index of 80 per cent for that week. He also later could be evaluated as having had a prodromal period since infancy but never had a light or intense phase, except for the week prior to perforation. This last patient was subjected a year later to subtotal gastrectomy because of increasing difficulty with pain and vomiting, and a lowering of his work index to 60 per cent or less for the two months prior to surgery.

Four patients were brought to surgery for relief of organic obstruction, and had light phases of 40, 14, 14, and 10 years, and intense phases of 4, 4, 2, and 2 years' duration, respectively. The first showed marked dehydration, and the last showed A/G reversal in his protein studies[8].

Of the three who were operated for bleeding, one illustrates the use of the index evaluation. He was a 22-year old male who had a work index of 4/24 (16.6 per cent) for 2 years, associated with three major episodes of bleeding, and only one of several repeated x-ray studies giving suggestive evidence of duodenal ulcer. There were several emotional episodes in this same 2-year period. Combined medical and psychiatric help had been ineffectual, even when 2 months hospitalization had been a part of the treatment. Following subtotal gastrectomy, and the initial 8-week recuperative period, this patient's work index was 100 per cent for the year for which he was followed. The emotional conflict situations were still a part of this patient's problems, but it seemed that bleeding had ceased to become a problem, and he was able to keep his place in society. Crohn interestingly discusses this problem of bleeding[16].

Of the three carcinoma patients, two had a known history of x-ray demonstration of duodenal ulcer by x-ray, 10 years' previously, and were over 60 years of age. The youngest had an intense phase of less than 2 months and at surgery showed a widespread metastatic involvement of his abdominal viscera. Two of these, including the last, or youngest, were of Holland Dutch ancestry, but this was not significant since the area was heavily populated with persons of Holland Dutch ancestry. There are, however, reports suggesting that perhaps dietary habits are responsible for increased incidence of gastric and esophageal carcinoma in the Holland Dutch and Chinese[17]. In view of the known genetic selectivity[18] of certain carcinoma strains, would it not be equally well to consider that these effects are the result perhaps of a genetic selectivity related to national existence. The United States, with its immense and various racial and national groups (both mixed and separate), allows an enormous so far untapped field for the study of human genetics in the field of gastric cancer. The same might be said for the natural and social factors of climate, region, water (content, limitation), food types, nearness to radioactive deposits, industrial hazards, and other environmental possibilities.

Three cases with light phases of 10 or more years showed serious protein disturbances when they finally submitted to medical help. Their only complaints had been increasing need for food restriction to relieve epigastric distress, and an associated weakness with marked weight loss. They responded to intense medical care.

It was noted of the 58 patients with a 2 or more years' intense phase history, that there was a great deal of disruption of the patient-doctor relationship. Two hundred sixteen changes occurred in these 58 cases during their intense phase histories. Seven, 6, and 14 individuals had changed doctors or clinics, 8, 5, and 4 times respectively. The patient often gave as his reason for seeking a change, the statement that the doctor had lost interest in the case. It was also indicated that there was a lack of symptomatic improvement. In this day of specialization, when we see cardiologists, diabetic specialists, vascular specialists, gynecologists, urologists, and other limited specialists, it is evident that we cannot claim any great number of doctors who may be considered limited gastroenterologists. It might be difficult to imagine what should constitute his field of work or training. It would seem that he should be well trained in the medical and surgical aspects of the gastrointestinal tract, yet be also trained in the applicable physiology, parasitology, psychiatry, and other problems that relate to this continuous organ. Whether or not such training would be justified would best be answered by the present state and national groups, societies, medical schools and other facilities concerned with serving the medical public. A unique consideration is offered by the American College of Gastroenterology, which invites all members of the medical and allied professions that might be able to contribute to the understanding of gastrointestinal disease to participate in its programs. In this way a balanced result is more likely than that which might follow the endeavors of a group limited to one aspect of the problem. Gastrointestinal, or gastroduodenal disorders are of themselves multifaceted and require multi-investigative measures.

Basic Similarities in Treatment Methods

If one looks broadly at the various methods of treatment in store for the gastroduodenal cripple, he can find certain similar fundamental principles. While not considered similar methods of treatment, surgery, internal medicine, psychiatry, and the patient's self-treatment may be said to all endeavor to quiet the patient's turbulent stomach. If the symptoms are not sufficiently disturbing to the patient, the treatment accordingly is not aggressive. In the quiet, lesser periods of distress, the patient or his physician restricts his diet to a compatible nutritive, nonirritating source of foodstuff. This does not mean that the food is at fault, it might more accurately be said that the stomach is at fault and cannot tolerate the foodstuffs ordinarily enjoyed by other human beings, or the patient when he is in the quiescent stage. Therefore, diet itself, or limitation to nonstimulating foods can be said to put the stomach at rest, and thereby aid in relieving the symptom-complex.

Effective psychiatric care aims at quieting the turbulence of the stomach, and actually seems to lessen the outpouring of the gastric juices. Rest cures can similarly be credited with lessening the stress and strain of mental problems. As these factors are accomplished, it is almost as a vicious cycle is broken, that allows the patient to again reach a quiescent stage, and avoid the complications, as we know them.

If acid is irritating, and we credit the neutralization of the gastric acids with healing of stomach ulcerations, then any method that lessens the amount or degree of acidity within the gastric cavity may actually be merely removing one of the aggravating factors in gastroduodenal disease. Surgical drainage, and antispasmodic drugs aid in removing the irritating juices from their site of action in ulcer production, if contact is a factor. Surgical excision or drug diminishment of the gastric acidity are accepted methods in the treatment of the patient with advanced peptic ulcer disease.

As to the obstructive element, it might be more correctly stated that the only true obstruction is the advanced, organic, stenosed, fibrotic obstruction that follows long lasting peptic ulcer disease. The accepted treatment for this is surgical relief, a mechanical drainage. It is interesting to note that all the surgical methods, that have found favor, have improved the drainage from the gastric hollow, be it by pyloroplasty, gastroenterostomy, gastrojejunostomy after subtotal resection, or as in vagotomy, these drainage methods have added to the value of the procedure[19]. Wangensteen has recognized the value of drainage in his excellent series of tubular resections, and describes the use of a proper pyloroplasty[20]. One might not be far amiss in also considering that a physiologic obstruction can be just as harmful to the body economy as the above mentioned organic obstruction. If the gastroduodenal segment does not effectively fulfill its function of passageway for food, it is not aiding the body nutritive state. Such physiologic obstruction can arise from spasm, local irritation, or it would seem, even from gastritis, if the patient suddenly finds that he cannot rely on his stomach to accept food placed therein.

If drainage from the gastric hollow is adequate, then the highly acid gastric contents are less in duration able to exert any direct effect on the gastroduodenal mucosa. Does it not then follow that any number of factors can contribute to a vicious cycle, which unless relieved in one way or another, can so aggravate itself that unless earlier evidenced by perforation, or penetration, or bleeding, can progress to eventual stenosis and true obstruction (Fig. 1)?

Would it be too far amiss then, to repeat what was said earlier, that all segments of the medical profession aim at the best restoration of the gastroduodenal cripple to a useful place in society, even though their methods may seem different?

Does it not seem that, in striving to find a single answer and explanation for there being such a cripple, we have been at times prone to forget that there

are apparently many factors, such as drainage, acidity, mucus deficiency[22], emotional reflex irritative patterns, and that we employ one avenue to the neglect of others?

In summary, we have merely tried to indicate that the gastroduodenal cripple may suffer acutely or chronically to various degrees, at any time in his life, and that there would seem to be many factors involved. We also feel that there is a long latent period before the usual case presents himself to a physician, and that this has not been sufficiently investigated. It is suggested that if such studies could be made on this group of individuals, earlier attention to selected "ill" patients might result in lessening the incidence of later complicated cases of peptic ulcer disease. It is also pointed out that starvation can be a complication of self-imposed or physician advised dietary restriction. The relationship of the gastroduodenal segment to the total body economy from the standpoint of a portal of entry of foodstuff is touched upon. The details of the effects of such a breakdown are left to the physiologists, endocrinologists, and nutrition experts. It seems plausible to assume that a vicious cycle underlies the progression of most cases of peptic ulcer disease, and that irritative and drainage factors are of prime importance in breaking this cycle. It is also mentioned that attention to the patient is as important, if not more important, than attention to his ulcer, and any treatment is ineffective if it does not contribute to the "portal of entry function" of the gastroduodenal segment. It is finally mentioned that there is a need for education of the patient and physician, and a correlation of all interested channels of activity for the eventual best care of these patients. This correlation can be in the individual physician's training or can be carried out in a hospital program or on a national level.

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VOLVULUS OF THE NEWBORN*

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Intestinal obstruction is a serious disease at any time, but the mortality curves reach their peaks at each end of the life span. In this paper we are particularly concerned with volvulus in the newborn which represents an interesting challenge to both pediatrician and surgeon alike. This subject has been presented by various authors[1-10] but most reports have not distinguished between volvulus in the newborn and volvulus in childhood, which although they may represent a similar pathological entity, vary greatly in survival rates and difficulties of management. Also the majority of papers have lumped together volvulus and malrotation of the mid-gut. These two entities though often in association with each other, are as a whole, quite different in severity and prognosis. While malrotation frequently is a chronic abnormality, many times unsuspected until discovered at laparotomy or on x-ray studies, volvulus usually is acute in onset and fulminating in course, carrying with it a corresponding severe morbidity and grave prognosis.

The material presented here is based on eight consecutive unselected cases operated on by the authors at the Margaret Hague Maternity Hospital, Jersey City, New Jersey, between October, 1947 through March, 1954. These operations were all carried out on newborn infants under 30 days of age and six of these were less than a week old at time of surgery. Seven of the eight cases presented "acute surgical abdomens".

Embryology

Because of the frequent co-existence of volvulus and malrotation, a review of the embryology of the intestines with an indication of possible danger points during the course of development is of value.

The gastrointestinal tract begins in the fetus as a straight, uniform tube suspended from the dorsal celomic cavity by a mesentery. The further progress of this simple structure to the typical adult pattern is conveniently divided into three phases[1,2,4,6,9,10]:

Stage I—5th to 10th fetal week:—The intestines grow faster than the abdominal wall, and a large portion of the mid-gut supplied by the superior mesenteric

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artery is forced outside the abdominal cavity and lies within the membranes of the umbilical cord. Arrest of progress at this stage results in an öomphalocele.

Stage II—10th to 11th week:—The mid-gut rotates 270° in a counterclockwise direction around the superior mesenteric artery as an axis, as it returns to the abdominal cavity with the relative faster growth of the abdominal walls. It is during this phase that most cases of volvulus of the newborn originate. Nonrotation here results in a bunching of the small intestines on the right side of the abdomen with the colon occupying the left half. A form of malrotation may occur in which the small bowel is reduced into its own mesentery with complete internal herniation, much the same as a fist is enclosed in a paper bag. Rarely a reversed rotation of 90° in a clockwise direction takes place in which the colon passes dorsally to the duodenum and the superior mesenteric artery.

Stage III—11th week on:—The intestines assume their normal adult position with fusion to the posteriolateral abdominal wall. Arrested progress at this point may result in abnormal fixation and location of the cecum, sometimes causing partial or complete obstruction of the duodenum. Occasionally a narrow fixation of the base of the small intestines develops with resultant predilection to volvulus of the entire small gut.

Symptoms

The diagnosis of volvulus of the newborn is not an easy one to make in many instances and requires alertness on the part of the nursing staff and doctors alike. Poor nursing of the infant, with regurgitation, may present the earliest symptoms. These of themselves are usually not considered serious signs but should alert the whole staff to possible future difficulties. Vomiting usually occurs early and is persistent. Most of the vomitus will contain bile in contradistinction to the vomiting of congenital hypertrophic pyloric stenosis, the higher duodenal atresias, and annular pancreas. Abdominal distention is always present and is greater, the lower the volvulus. This is often a late manifestation and in high volvulus may be largely due to generalized peritonitis. Careful observation of the stool is of great importance. Lack of bowel movements in infants is always a sign of trouble, however, a child may be obstructed from birth and yet pass a few meconium stools, particularly if the twist is in the upper jejunum.

Physical Findings

Because of vomiting, all patients will show signs of dehydration unless fluid balance has been maintained by supplemental measures. Some degree of abdominal distention is almost always present, being absent only in the early high obstructions. When distention exists in only moderate degree, abdominal rigidity is usually not present, bowel sounds frequently are hyperactive and associated with borborygmi, and on occasion, a definite mass can be palpated in an area

TABLE I

EIGHT CASES OF VOLVULUS OF THE NEWBORN

Name	Sex	Onset of Symptoms From Birth	X-ray Evidence	Age at Operation	Location of Volvulus	Gangrene	Type of Operation & Date	Nil per os postop.	Survival
1. Simone 57755	F	2 days	gas & fluid levels	3 days	Terminal Ileum	No	Reduction 9/3/47	2 days	Yes
2. Hempel 75324	F	2 days	gas & fluid levels	6 days	Upper Ileum	Yes	Resection with side-to-side anastomosis 12/14/47	3 days	Yes
3. Pilnick 102482	M	0 days	gas & fluid levels	2 days	Lower jejunum	No	Reduction 5/14/50	3 days	Yes
4. Strickland 102408	M	2 days	gas & fluid levels	3 days	Mid small bowel	No	Reduction 5/14/50	4 days	Yes
5. Veronis 126652	M	1 day	gas only	4 days	Mid small bowel	No	Reduction 8/6/52	2 days	No*
6. Valle 134881	M	8 days	contrast medium puddled at Lig. of Treitz	29 days	Upper jejunum	No	Reduction 5/23/53	3 days	Yes
7. Singleton 137189	M	19 days	gas only	23 days	Terminal Ileum	Yes with perforation	Resection with-end-to-end anastomosis 7/26/53	5 days	Yes
8. Nahaczewski 144333	M	2 days	gas & fluid levels	3 days	Jejunum, with megacolon	No	Reduction 3/15/54	3 days	Yes

All cases were operated upon under open ether anesthesia.
*See footnote Table VII

of the abdomen comparable to the level of the twist. In greatly distended abdomens, rigidity or muscle guarding is more of a factor, auscultation reveals a relatively silent abdomen, and a mass is much less likely to be felt. Tenderness and especially localized tenderness, is frequently difficult to elicit except when accompanied by a mass or peritonitis. Rectal or bimanual examination may be of value in establishing a diagnosis when the volvulus is located in the ileum.

X-RAYS

The use of x-rays in the preoperative diagnosis of volvulus is of great value, however, many pitfalls lurk for the unwary and the interpretation of adult and infant plates differ in certain aspects. All cases of volvulus will show gaseous distention of some portion of the small bowel. High obstruction may only distend one or two loops of jejunum in the left upper quadrant of the abdomen, while a twist of the terminal ileum will create enlargement of the whole small bowel. Fluid levels are significant when present but are usually a late finding and often are not found preoperatively; to wait for them to appear can prove disastrous. A disparity in the size of the visualized loops of small bowel is the most constant and significant x-ray evidence of obstruction.

TABLE II

SEX INCIDENCE

Males	6 cases
Females	2 cases

Flat plates are not sufficient and should be supplemented by uprights and right and left laterals. Serial x-rays may be used in particularly cryptic cases with the aid of lipiodal injected into the stomach through a Levin tube. Barium is best avoided, as aspiration can cause serious pulmonary difficulties.

PREOPERATIVE PREPARATION

Once the diagnosis has been established, or the decision to operate has been made, no time should be lost in getting the infant ready for the operating room. Particularly is this so as often many hours have elapsed from the onset of symptoms and it is imperative to get the patient to surgery before irreversible changes in the bowel have developed, as resection with anastomosis increases the mortality rate sharply. On the other hand a few hours used in preparing the infant for laparotomy may change a moribund case into one of reasonable risk.

Infants of this age group tolerate dehydration very poorly and every effort should be made to correct fluid and electrolyte balance preoperatively. A Levin tube should be passed into the stomach and its content aspirated. This will help deflate the bowel, making anesthesia safer, and will prevent the aspiration of

vomitus with its resultant atelectasis and pneumonia. A warm tap water enema may aid in elimination of gas and stool from the colon and if given with discretion should cause no harm to the small intestines. A cut down in an ankle vein with insertion of a fine polyethylene tube should be performed for free access to the vascular system during surgery and for postoperative therapy.

Anesthesia

Although local anesthesia has been advocated in the past, it is generally agreed that it is inadequate due to the abdominal distention always present. Light open drop ether is the method of choice, but where trained anesthetists and equipment are available, a closed or partially closed system using a Digby-Lee valve or some similar attachment, may be employed to advantage. Where meconium ileus may also be present[5], cyclopropane is indicated to avoid the irritating qualities of ether in the respiratory system. It is much better to err on the side of too light anesthesia than the reverse, as the safety factor in these young gravely ill patients is small and its borders ill-defined. Oxygen should be administered in copious amounts and a free air-way maintained at all times.

TABLE III

Onset of Symptoms from Birth

Cases	1	1	4	1	1
Days	0	1	2	8	19

Operation

The infant is best managed by being secured to a padded frame and should be protected from heat loss by warm water bottles and blankets.

Most surgeons prefer a split rectus or paramedian incision with lateral retraction of the rectus muscle. This allows for facility in performance, ease in extention, free access to all parts of the abdomen, and rapid closure.

Many cases of volvulus can be cured by simply untwisting the involved segment of gut. Reduction of volvulus within the abdominal cavity was found to be impossible in most cases where the segment of bowel involved was more than a single loop of intestine. Appreciating the trauma associated with the exposure of long lengths of bowel, none the less, experience points to the absolute necessity of immediately eviscerating the whole portion of the involved intestine on the surface of the abdomen when the volvulus is extensive. The twisting is more easily corrected, the bowel is replaced with gentle pressure applied through the aid of warm, moist lap pads and sponge sticks. Rapid reduction of the volvulus by evisceration seemed to be a mandatory clinical procedure in our hands in most cases. This entire procedure should occupy only a few minutes. No delay can be tolerated with these labile patients as it increases the chance of develop-

ing shock and prolongs the duration of anesthesia. Every effort should be made to avoid resection of the intestines or any type of by-passing anastomosis because of their inherent elevation of the mortality statistics. Ladd's[7,8] contribution to malrotation is that he recognized concurrent duodenal encroachment by ligamentous bands in a high percentage of cases and proposed a liberating operation which obviated an enteroanastomosis, hence causing a reduction in the previously published mortality figures.

When necrosis has proceeded so that resection is mandatory, practically any type of anastomosis is adequate, and an end-to-end re-establishment of bowel is reasonably easy if interrupted sutures of fine caliber are used and gentleness

TABLE IV

AGE AT OPERATION

Cases	1	3	1	1	1	1
Days	2	3	4	6	23	29

practiced in the handling of tissues. Enterostomies in the small intestines after the manner of Witzel, should be avoided as they function poorly and often add to the postoperative complications.

The abdominal wall should be closed with a minimum of buried catgut which interferes with wound healing and is readily disintegrated by the infant's increased metabolic processes. The fascia is best approximated by interrupted sutures of nonabsorbable material, which may be left in place or used as figure-of-eight retention sutures for fascia and skin combined.

TABLE V

X-RAY EVIDENCE OF OBSTRUCTION

Increased air	7 cases
Fluid levels	5 cases

POSTOPERATIVE CARE

No phase of treatment is more important in bringing about the survival of these patients than the postoperative management. To operate and then slacken up, is to court disaster. Alert nursing and house staff attention is often the deciding factor in recovery.

The respiratory passages of the patient should be cleared by suction prior to leaving the operating room and must be maintained in that condition throughout. Body heat must be preserved and oxygen supplied in adequate amounts, both of which may be regulated nicely by placing the patient in some type of incubator or isolette. Nil per os and gastric suction should be continued until active peristalsis returns and bowel movements are established. Fluid require-

ments, calculated on a weight basis, must be supplied totally or partially until the infant can take enough into its gastrointestinal tract to maintain proper fluid balance. Blood studies will aid in the decision as to possible blood transfusions. Antibiotic therapy is of importance regardless of whether peritonitis was present or resection was performed, as it will help in the prevention of postoperative lung and wound complications. If nonabsorbable sutures have been used to approximate the skin, studied delay in their removal may keep a dehiscence from becoming an evisceration.

Case Report

Infant Singleton (Case 7, Table I), a Negro male, was born on 7/3/53 and discharged 7 days postpartum with what appeared to be normal physiological icterus. The patient was re-admitted 17 days postpartum with a history of ten days of chalky stools and dark yellow urine, eating well, no nausea or vomiting, and a temperature of 102.6°. Admitting diagnoses were: Infectious Hepatitis or Septicemia.

The temperature remained elevated between 102-3° for five days. The abdomen began to distend and x-rays were taken on the 21st and 22nd pospartum

TABLE VI

Nil Per Os Postoperatively

Cases	2	4	1	1
Days p.o.	2	3	4	5

days showing uniform gaseous distention of the whole small bowel but no fluid levels, and exhibiting the general appearance of a paralytic ileus. Surgical consultation was sought. There was still no vomiting although no bowel movement was recorded for the previous 72 hours. The abdomen was silent, distended and nontender. There was edema of the lower anterior abdominal wall. The temperature was 102°. Deep jaundice was present and all tests showed it to be of the obstructive type. Supportive measures were begun immediately. Next morning the temperature was 101.8°, the abdomen was more distended, no bowel movements had occurred in the interim, and rectal irrigations returned only bloody tinged mucus.

A laparotomy was performed through a split right rectus incision under open ether. A small segment of terminal ileum was twisted, gangrenous, and perforated; being adherent to the base of its own mesentery. The gallbladder was distended with green bile. The involved segment was resected and an end-to-end anastomosis made. The abdomen was flushed out with warm saline and a Penrose drain placed in the pelvis and brought out through a stab wound lateral to the incision. The abdominal wall was closed in layers with black silk retention sutures for skin and fascia. The child was returned to the ward, placed

in an isolette, and the usual postoperative treatment instituted, including Levin tube suction, nil per os, antibiotics, vitamins and oxygen. Intravenous fluids, protein-hydrolysates, and blood transfusions were administered through a polyethylene tube placed in an ankle vein preoperatively. The first bowel movement occurred 48 hours after surgery. Oral feedings were begun on the 5th postoperative day. On the 8th postoperative day an evisceration of the lower angle of the wound was discovered. This was resutured with through and through sutures of black silk and the remainder of the postoperative course was uneventful.

The patient remained in the hospital for further supportive therapy and investigation. Jaundice of the obstructive type persisted. Fifty-nine days from the original operation a second laparotomy was done under open ether using a subcostal incision. An atresia of the retroduodenal portion of the common duct was found and a cholecystoduodenostomy performed. The postoperative course was smooth and follow-ups in clinic have shown a complete clearing of jaundice, a return of serum bilirubin to normal values and a concurrent weight gain, now a year postoperatively.

TABLE VII

SURVIVAL

Survived	7 cases
Expired	1 case*
Rate Survival	87.5%

*This case, #5 in Table I, expired on the 4th postoperative day of uremia from anuria. Autopsy diagnoses included mongolism, malrotation of hind gut, bilateral hydronephrosis with urethral obstruction, bilateral atelectasis, patent ductus arteriosis, patent foramen ovale and plastic peritonitis. The volvulus present at operation did not require resection, and remained corrected during the postoperative course, as several stools were recorded right up to the time of exodus.

COMMENT

Volvulus in the newborn is usually an acute and fulminating disease leading to death if untreated or diagnosed too late. Because of the acuteness of the condition it is imperative to make an early diagnosis, get the patient ready for surgery, and operate before gangrene occurs in order to maintain a high salvage rate. To further this end everyone coming in contact with newborns should be on guard for the symptoms and signs of this disease. It is better to get a surgical consultation *before* the diagnosis is solidly established as every surgeon is familiar with the advantages of a "second look" at an acute abdomen, especially in a patient only a few hours old. Valuable time may be saved by this simple procedure, and by the same token, the mortality rate will be lowered.

Seventy-five per cent of our cases developed symptoms and were operated upon during the first week of life. The usual sex distribution occurred with 75 per cent of the cases being males.

X-rays were of great assistance in making a diagnosis, being positive in all cases. To wait for fluid levels to develop, however, is to court disaster. One of our patients (Case 7, Table I) did not show fluid although gangrene with perforation of the terminal ileum was found at laparotomy. It is also important for the surgeon to review the x-rays personally rather than accept a written report from a radiologist who has not seen the patient and who may not have had much experience in reading infants' x-rays. Especially is this so in smaller institutions.

Although we are cognizant of Ladd's[7,8] contribution to the surgical correction of malrotation in infants, the frequency of duodenal obstruction in acute volvulus of the newborn is open to question in spite of the stated association of these entities, for in none of our cases were we able to demonstrate infringement on the lumen of the duodenum by congenital bands due to malrotation or nondescent of the cecum.

Expert anesthesia is of great value when available. These patients have a lower physiological reserve and the limits of error are narrowed. We do not feel that children below six months of age should have elective surgery for this reason and if emergency operations are necessary, every effort should be made to secure top notch anesthesia and to observe all safeguards during its administration. In many hospitals these patients receive less than the best available anesthesia because of a lack of interest in this type of case. It is here that special pediatric hospitals with their anesthetic staffs, have an advantage over the general hospital where the giving of infant's anesthesia is often relegated to a less experienced individual.

Unless a surgeon has mastered the art of gentleness in the handling of tissues and reasonable surgical dexterity in performing an operation, he should allow some one else to do these cases, for roughness, poor technic, and unnecessarily prolonged operating time are all great deterrents to the survival of these gravely ill patients.

While it is advantageous to establish oral feedings as soon as possible during the postoperative period, caution must be exerted to retard the natural tendency of all personnel to reach for the nursing bottle. The return of active peristalsis, bowel movements, and a decrease in abdominal distention should occur before allowing oral alimentation. From Table VI it can be seen that usually this happened on the 3rd postoperative day in our cases. We have no hesitation in using clyses and in one infant operated on by the senior author (K. J.), for a different condition, fluids were not tolerated until the 28th day postoperatively, yet the patient recovered. Part of the success of these clyses may be due to the fact that after calculating the amount of fluids required in a 24 hour period, half the total is given in Ringer's Solution, and half in 3½ per cent Amigen.

The survival rate for this small series of cases as charted in Table VII has been exceedingly good, and here again let us stress the fact that figures on

simple malrotation in children should not be compared with the statistics on acute volvulus of the newborn. The part played by the consulting pediatricians cannot be over-evaluated. Gross[3] has ventured the opinion that every surgeon who operates on these cases should carry out all the pre- and postoperative care also While we concur to some extent with this hypothesis, and certainly feel that the surgeon should not limit himself to the role of technician exclusively, still in practice, the close cooperation of the pediatrician and surgeon working as a unified team, will in all probability result in the most optimum care for the patient. Again may we state, that the role of the pediatrician in the above survival statistics has been invaluable.

Summary

1. Eight cases of volvulus of the newborn have been presented. One death occurred on the 4th postoperative day from a number of congenital malformations as reported in the postmortem findings.

2. The differences between malrotation of the intestines in children and acute volvulus in the newborn, have been stressed, both as to management and prognosis.

3. It appears that if the mortality rate is to be lowered, all personnel handling newborns must be alert for the first symptoms of this disease and early surgical consultation obtained, preferably *before* the diagnosis has been definitely established.

4. The team work of pediatrician and surgeon along the whole course of the illness is of inestimable value in maintaining a high survival.

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THE PANCREATIC SECRETIN TEST AS A DIAGNOSTIC AID*

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We have used the pancreatic secretin test as an aid in diagnosis of patients with bizarre abdominal complaints. From the work of Bayless and Starling[1] we know that pancreatic juice and bile are stimulated by action of secretin, a duodenal mucosal hormone. Clinical use of this substance is practical since it is a commercially produced, standardized product† and since uncontaminated duodenal fluids can be obtained by use of the double lumen gastroduodenal tube described by Lagerlof[7].

Secretin stimulates the flow of water and bicarbonate from the pancreas. Pancreatic enzymes are washed out with the increased volume of water, and as a result the concentration of enzymes may be less significant than the amount of bicarbonate.

Although secretin stimulates bile production by the liver, bile is increased in the duodenal juice only when the gallbladder capacity has been overreached, or when the gallbladder has been removed or the cystic duct occluded.

For purposes of differentiation, a biliary pigment curve has been devised by Diamond and Siegel[3] based on determination of the icteric index of duodenal fluid collected before and after secretin stimulation. The normal curve has a falling icteric index approximating zero. In the presence of a nonfunctioning or absent gallbladder, the curve shows a constant increase in concentration of bile pigment. A third type of curve occurs when bile cannot enter the duodenum and the duodenal fluid remains clear throughout the test.

Thirty-one patients clinically suspected of pancreatic dysfunction were tested in the following manner. Sitting in an upright position, the fasting patient swallowed a double lumen gastroduodenal tube with a mercury bag attached to the end. Whenever possible the tube was inserted the evening prior to the test period to insure entrance into the duodenum. It was possible, however, to do the test on out-patients by inserting the tube early in the morning. The tube was then attached to a Wangensteen suction apparatus and the gastric and

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†Wyeth Chemical Co.

duodenal aspirations collected separately. If the duodenal aspirations were alkaline, in the presence of normally acidic gastric juice, it was assumed the tube was in the duodenum. Whenever there was uncertainty concerning the position of the tube, the patient was fluoroscoped. Fasting samples of duodenal juice and blood were collected and secretin (usually one unit per kilogram body weight) was given intravenously. The next three specimens of both duodenal fluid and blood were taken at 10, 20, and 30 minute intervals after secretin injection. Those specimens of duodenal fluid were discarded in which the pH was below 7, because of probable contamination with gastric fluid.

The duodenal fluid was analyzed for pH, bicarbonate level (titration against N/10 hydrochloric acid), amylase (modified Somogyi method, normal fasting

TABLE I

NORMAL RESULTS WITH CONFIRMED LESIONS

Case No.	Surgical Procedure and Diagnosis
3	Cholecystectomy for adenocarcinoma of the gallbladder
4	Partial resection left lobe of liver for biliary retention cyst
10	Cholecystectomy for chronic cholecystitis with cholelithiasis
19	Abdominal perineal resection for adenocarcinoma of the rectum
24	Cholecystectomy for chronic cholecystitis with cholelithiasis
29	Lysis of adhesion about liver and duodenum at previous cholecystectomy site
30	Laparotomy with resection of lipoma of the mesentery
31	Right salpingectomy for pyosalpinx
32	Resection of the common bile duct tumor with an end-to-end anastomosis for leiomyofibrosarcoma of the common bile duct

1,500-2,500 u. per 100 ml. of fluid), lipase by action on ethyl butyrate (normal fasting 2-4 u. for 100 ml. of fluid), and icteric index.

The blood serum was analyzed for amylase (Somogyi, normal 50-150 u. per 100 ml.), and lipase (normal 0.2-2.0 u. per 100 ml.). The results are considered abnormal in samples with values above or below these accepted normal levels or in those with no response to secretin stimulation.

Thirty-one patients were examined according to the procedure outlined above. Of these patients, 18 had normal test results and 13 had abnormal test results. In eight of the 18 patients with normal results no pancreatic lesions were found during abdominal exploration. The surgical diagnosis of each patient is listed in Table I.

A ninth case in this group had a normal response to secretin stimulation except for an absence of bile in the duodenal fluid. This finding indicated a block of the common bile duct above its junction with the pancreatic duct. This was confirmed at operation (Table I, Case 32). The other nine patients with normal test results were placed on medical management. On a fat free diet and cholagogues, after three years, six of the patients are doing well and one patient has had no improvement. It is doubtful if this patient adheres to the treatment suggested. The eighth patient was treated for hypoglycemia with relief of symptoms. In the final patient further investigation has led us to believe that her symptoms are psychic in origin. Two subsequent attempts to repeat the test have failed because we could not pass the tube into the duodenum. It is quite possible that this patient has a spasm of the sphincter of Oddi as a result of emotional disturbance.

TABLE II

ABNORMAL RESULTS WITH CONFIRMED LESIONS

Case No.	Lesion Found at Operation
5	Partial obstruction of the pylorus due to chronic duodenal ulcer
18	Pancreatitis associated with common duct stone
22	Pancreatic fibrosis
34	Duodenal ulcer with fibrosis and edema of the head of the pancreas
	Lesion Found at Necropsy
15	Carcinoma of the head of the pancreas
9	Fatty infiltration of the pancreas
11	Carcinoma of the pancreas

Of the 13 patients with abnormal test results seven were found to have pancreatic lesions at operation or necropsy.

The test was repeated on Case 18 six weeks postoperatively and the results were still abnormal. Three months later, however, the test results were normal and the patient was clinically well.

In Table III are summarized the findings of the remaining six patients in the group with abnormal test results, the abnormal response occurred in one patient (Case 25) after the third evisceration following a vagotomy and gastroenterostomy at which no pancreatic lesion was found. This patient was given no specific treatment but improved clinically and is well two years later. Two patients improved on diet and antibiotic therapy. One patient has refused to follow a prescribed diet or medication and has not improved. Two patients subsequently expired at home and permission for necropsy examination was not granted (Cases 21 and 33).

Comment

In the performance of the secretin stimulation test Lagerlof[7] has recommended a test period of 80 minutes, and Diamond[4] a test period of 60 minutes which corresponds to our own method. Dornberger[5], however, has recommended shortening the test period still further out of consideration for the patient and because the maximum effect of the secretin is reached in 40 minutes. In acute pancreatitis the secretin test is probably of very limited value (Dreiling and Hollander[6]). In those patients with an elevated serum amylase level during the acute attack it is possible that the pancreas retains a considerable amount of functional tissue, thus the fluid secreted has a normal volume but a relatively low enzyme content.

During the first week following an attack of acute pancreatitis the test is very difficult to perform. During the second week even though clinical symptoms persist, the function of the pancreas may be so restored that the results of the

TABLE III

Abnormal Results with Unconfirmed Lesions

Case No.	Results
21	Expired at home (no necropsy) Idiopathic Sprue?
25	Improved–Vagotomy and gastroenterostomy
27	Not improved, did not stay on diet or medication
33	Slightly improved at first, expired at home (no necropsy)
35	Improved on high protein, carbohydrate and low fat diet
37	Improved on antibiotics and low fat diet

test are normal and in some individuals there may be a moderate impairment of function with normal enzyme levels and only slightly lowered volume. At operation these patients may have residual edema, swelling and eventual fibrosis with severe or progressive damage to the pancreas. The function as demonstrated by this test progressively declines.

In patients with severe fibrosis and in those with calcification there is usually an abnormal test result. In these patients there may be a considerable depression in bicarbonate value and in the enzyme content.

In chronic pancreatitis Comfort[2] has found with secretin and mecholyl stimulation an abnormal increase in volume with a hyposecretion of amylase and a hypersecretion of proteinase and lipase. In those tumors of the pancreas with obstruction of the pancreatic ducts there is a reduction in the volume of pancreatic juice but few other significant changes.

In patients with common bile duct obstruction there will be little if any bile in the duodenal fluid. In some of our patients with chronic cholecystitis there was a diminution in secretion even though in none was there evidence of a primary pancreatic lesion.

Summary

1. The secretin stimulation test of pancreatic function was performed on 31 patients.

2. The technic of duodenal fluid aspiration and test methods are described.

3. In our hands this test has been an aid in the diagnosis of pancreatic lesions as a result of chronic pancreatitis with or without fibrosis and calcification, and in the recovery phase of acute pancreatitis.

4. This test has also been an aid in the diagnosis of common bile duct obstruction.

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COMPLETE HEALING OF PEPTIC ULCER WITH WATER SOLUBLE CHLOROPHYLL

Report of a Case

W. G. OFFENKRANTZ, M.D.

Brooklyn, N. Y.

Although the management of peptic ulcer involves consideration of complex etiological factors, the desired end-result is complete healing of the ulcer crater. Specific therapy for accelerating the healing process, therefore, will play an important role in peptic ulcer cases.

The use of antacids in neutralizing the action of hydrochloric acid and of demulcents in coating and protecting the ulcer crater is well known. That these agents are helpful has been abundantly shown. Yet the healing process may be

Fig. 1—Patient M.B., December 2, 1951.

disappointingly slow and incomplete with recurrence a likely possibility in many instances. It is logical to assume, therefore, that the addition of a tissue-stimulant to antacids and demulcents would assist the damaged gastroduodenal tissue in making a complete recovery.

Laboratory and clinical studies have shown that water-soluble chlorophyll possesses tissue-stimulating properties. In a series of 1,372 animals with experimentally-induced wounds, Smith and Livingston[1,2] found that chlorophyll reduced the healing period by 25 per cent in more than two-thirds of the group as compared with controls. Rapp and Gurney[3] demonstrated that chlorophyll

inhibits the action of proteolytic bacteria and enzymes. At the Lahey Clinic, Boehme[4] reported on the high percentage of rapid healing in 50 cases of chronic leg ulcers by the use of chlorophyll. This action of chlorophyll has been confirmed by subsequent studies on chronic wounds, burns and dermatoses[5-7].

In a recent preliminary report on 27 cases of gastric and duodenal ulcers, Offenkrantz[8] obtained symptomatic relief and complete healing of the ulcer crater in 20 cases as shown by roentgenologic examination. The average length of treatment was four weeks. Previous ulcer therapy for this group covered periods of from 2 to 12 years.

A dramatic complete peptic ulcer healing in a recent case prompts this report since it highlights the remarkable efficacy of chlorophyll. The chlorophyll powder used consisted of water-soluble chlorophyll, aluminum hydroxide, magnesium trisilicate and gastric mucin. The method of treatment was as follows:

Fig. 2—Same patient, March 8, 1952.

Five chlorophyll powders per day were prescribed—one upon arising, one upon retiring, and one after each meal (one and one-half hours after meal). Patient was instructed that no other fluids or foods should be taken at least one and one-half hours after powder. The patient's normal habits as regards smoking and alcoholic beverages were not restricted, except that the consumption of alcoholic beverages, like other fluids, was confined to meal time and not before one and one-half hours after taking of powder.

Case Report

M. B., a 38-year old male, was first examined on December 8, 1951. At that time he complained of precordial pain accompanied by epigastric pain radiating to the back. There was no night pain, nausea, vomiting or tarry stools. The pain

was relieved, the patient stated, by frequent intake of food, especially hot milk. Smoking had been cut to half his former amount and there was no consumption of alcoholic beverages. His past medical history was negative except for an inguinal herniotomy many years ago. He was on a bland ulcer diet and oftentimes restricted himself to only milk and toast.

The patient was employed as a production manager in the dress business which involved extremely great tension and aggravation. He worked in Vermont and came home to New York on weekends.

Because of his pain, the patient had previously consulted various other physicians. An x-ray taken on December 2, 1951, by one of the physicians revealed a small orthotonic stomach of the fish hook type with a large ulcer crater about ½ inch in diameter on the lesser curvature approximately 2 inches above

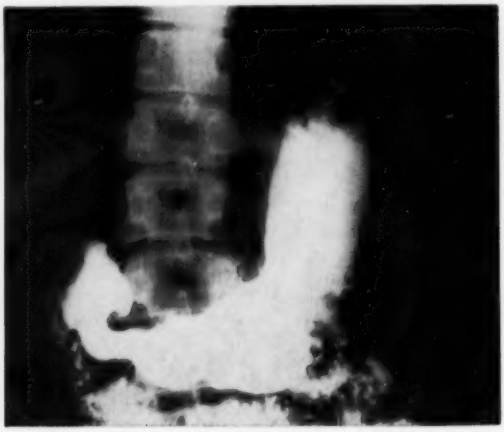

Fig. 3—Same patient, October 29, 1953.

the incisura angularis (see Fig. 1). The colon was normal and evacuation unimpeded.

In view of these x-ray findings, surgery was advised by these physicians as well as by the author. The patient, however, was deathly afraid of surgery and refused. Consequently, the patient was put on the chlorophyll powder regime. No attempt was made to restrict the patient's diet in any way. He was permitted to eat whatever he wanted. There was no restriction to his smoking. The patient's occupation permitted no physical or mental rest.

After one week of chlorophyll powder therapy, the precordial and epigastric pain had almost completely disappeared. On the third week there was no pain and the patient felt fine, "eating everything" with no untoward consequences. On March 8, 1952, a little more than 3 months after his first x-ray pictures and

3 months after initiation of chlorophyll therapy, x-rays were taken (see Fig. 2). There was complete absence of the large penetrating ulcer previously present on the lesser curvature. Follow-up x-rays on October 29, 1953, more than one year and seven months later, showed no evidence of recurrence although the patient's work habits remained the same.

Summary

A report of this case of peptic ulcer has been presented in which complete prompt healing took place following administration of chlorophyll powder. There was no recurrence, as evidenced by roentgenologic examination, more than 1½ years later.

It should be noted that no ulcer regime was followed; that smoking was not prohibited; the patient's tension and way of life still continued.

It is felt that chlorophyll powder acts as an efficient tissue-stimulant so that complete ulcer healing takes place, reducing the incidence of recurrence. Together with antacids, a further study of chlorophyll powder in the management of peptic ulcer appears warranted.

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In Memoriam

We record with profound sorrow the passing of Dr. Sylvan Strong Scholpp, Member, of Phoenix, Ariz. We extend our deepest sympathies to the bereaved family.

President's Message

Your President has just returned from a conference with your Secretary, Dr. Joseph Shaiken, Dr. Michael W. Shutkin in charge of Scientific Exhibits, Dr. Edward J. Krol in charge of Arrangements, and Daniel Weiss your Executive Secretary. We met at our convention hotel, The Shoreland, 55th and South Shore Drive, in Chicago. The purpose was to perfect the plans, and work out a smooth running schedule of events for our convention, the week of October 23rd.

Mrs. Ferguson, Mrs. Shaiken, and Mrs. Krol were present in the interests of the Program for the Women's Auxiliary.

The hotel is rapidly being altered, and geared, to fit the needs of a Convention Assembly. The location, overlooking Lake Michigan, and physical plant are ideal for such a project. It is spacious, and regal in its atmosphere, quiet, pleasant, and home-like. It is well away from the noise and confusion incident to the down-town area. The loop is easily accessible in 10 minutes by way of the Illinois Central Railway. Busses, and cabs are available at all times, in sufficient quantities. Those preferring their own cars, will find ample and convenient parking space.

Mrs. Margery Bellows, the Managing Director of the Shoreland, is a most charming and cooperative person. She has caught the key-note to hospitality, and finds it easy to induce her personnel to reflect the same attitude. She is an Executive who sets an example and, through respect, her people follow suit. Success is hers, because she adheres to the simple policies based on the Golden Rule.

A few innovations are being tried out this year and, of necessity, they create new problems. Committee meetings will be held on Saturday, preceding the Convention, and the Convocation will be held on Sunday. Have your secretary block out your time, and get your hotel reservation in early, in order that you may have the accommodations that you like. Come prepared with suggestions, and the initiative to help work them out. Your College needs concerted effort, and good team-work all along the line.

The Programs are all in order, and many excellent speakers will be on the job in the interests of good Gastroenterology. "See you in Chicago".

Lyon A. Ferguson

ABSTRACTS FOR GASTROENTEROLOGISTS

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STOMACH

PRINCIPLES OF PEPTIC ULCER THERAPY: James F. Crenshaw: Clin. Med. 2:249 (Mar.), 1955.

In peptic ulceration the primary purpose is to protect the mucosa from hydrochloric acid. Excessive secretion is generally considered to be the cause. In duodenal lesions, hypersecretion is causative, while in gastric ulcers reduced tissue resistance seems to be contributory.

Hyperacidity should be controlled and the general well-being of the patient promoted. Physical and mental trauma, dietetic control, and avoidance of alcohol and tobacco favor recovery. Usually the patient is maladjusted so that "reeducation" is indicated. The cause of tension in the home, business and social life should be removed. Psychotherapy plays a significant role in the treatment of peptic ulcers. Generally the family doctor can provide the necessary psychotherapy.

The dietetic program graduates from hourly milk-cream mixtures up to three bland meals with supplemental feedings according to the conditions.

Antispasmodic of choice: Atropine Sulfate. Of the newer antisecretory compounds Pro-Banthine, Pamine, or Antrenyl seem to be satisfactory.

Calcium carbonate is an effective antacid; in two to four grams every hour or two. Aluminum hydroxide two grams every two hours is also useful. In refactory cases continuous drip may be valuable.

Surgery is indicated only if prolonged medical care is futile.

Thirteen to 20 per cent of patients thought to have benign lesions were found to be malignant. Medical treatment should be continued only if there is progressive diminution of the ulcer.

Resection should be considered in: lesions doubtful as to cancer, persistence after therapy, recurrent hemorrhage, ulcers of greater curvature, delayed emptying.

Perforation and obstruction are the main complications. Perforation requires immediate surgery. Bleeding requires hospitalization; but in absence of vomiting, feeding and antacid therapy is indicated. Transfusion should be available. Intravenous feedings are practiced in vomiting.

REGINALD B. WEILER

THE CHIEF MEDICAL PROBLEM IN MILITARY MEDICINE: PEPTIC ULCER; MEDICAL ASPECTS: John H. Willard. Military Med. 116:179 (Mar.), 1955.

Displacing infectious hepatitis, ulcer has become the chief medical problem in the European area—representing as high as 10 per cent the medical hospital admissions. Recurrences and complications with subsequent rehospitalization have become the rule rather than the exception. Positive x-ray findings in the European Theatre are reported as high as 60 per cent in contradistinction to about a 20 per cent positive radiologic diagnosis in routine gastrointestinal x-rays in the U.S.A.

Comparison of the relative incidence of peptic ulcer in civilians and service person-

nel does not suggest any great disparity. The high hospital admission rate apparently indicates frequent recurrences of symptoms in a group of men who are poorly motivated or who have been poorly integrated into the service. To achieve a satisfactory program to provide adequate treatment and prevent recurrences the author recommends:

1) Decreased stress, vacation, hospitalization and sedation.

2) Gastric rest: hourly feedings or at least frequent bland feedings;

3) Avoidance of stimulants: tobacco, caffein, alcohol and condiments;

4) Medication: antispasmodics and anticholinergics, antacids and demulcents;

5) Education and prolonged follow-up to prevent recurrences.

LOUIS A. ROSENBLUM

INTESTINES

VOLVULUS OF THE TRANSVERSE COLON: L. S. Figiel, and S. J. Figiel. Radiology 63:832-835 (Dec.), 1954.

An interesting case is reported of a volvulus of the transverse colon which occurred as a result of herniation through a tear in the transverse mesocolon. Except for the uncommon position, the radiographic studies of this type of mechanical obstruction are the same as elsewhere.

IRVIN DEUTSCH

PRIMARY TUMORS OF THE SMALL BOWEL: T. A. Ogilvie and H. M. Shaw. Brit. M. J. 4906:142 (15 Jan.), 1955.

The authors report a series of 16 cases of primary tumors of the small intestines treated at their hospital, Essex County Hospital, Colchester, England. Among these were adenocarcinomas, sarcomas and carcinoids.

The presenting symptoms were usually those of small bowel obstruction, and occasionally melena.

The carcinomas were all associated with poor prognosis even after surgical resection of the lesions.

The lymphosarcomas were poorly prognosticated.

The carcinoids most common in the appendix and terminal ileum were discussed particularly with reference to metastases. Those found in the appendix metastasize in only 5 per cent of instances, while metastases in ileal carcinoids is more frequent and more extensive.

H. M. ROBINSON

ZUR PSYCHOTHERAPIE DER COLITIS ULCEROSA: F. Curtius and H. G. Rohrmoser. Deutsche med. Wchnschr. 80:105 (21 Jan.), 1955.

The authors state that the exact etiology of ulcerative colitis is still unknown. Medicine, surgery and psychotherapy are competing in tackling the therapeutic difficulties which this disease presents. Neither antibiotics alone and/or in combination with ACTH and Cortisone did effect a cure per se. These medications are very valuable only to combat complications. The same therapeutic value is attributed to surgery. Of late psychological facts explaining the underlying reasons of ulcerative colitis have come more and more to the foreground. Different psychotherapeutic methods have been recommended.

Clinical experience shows that many patients with ulcerative colitis represent a neurotic personality.

Long lasting stress leads to chronic irritation of the colonic mucosa. Simultaneous lowering of the resistance of the mucosa and secondary bacterial invasion produce the full picture of ulcerative colitis.

A prerequisite for any psychotherapy is a close patient-physician relationship. A thorough understanding of the patient's personality is essential. Psychoanalysis leads the way towards further psychotherapeutic measures. Very often patients are too sick to use this time consuming method. Therefore, in order to save time, the authors apply hypnosis without detailed analysis. This way they get immediate complete mental and physical relaxation of the patient. The authors consider this relaxation of utmost importance to achieve a cure.

Four cases of ulcerative colitis are described in detail. These four cases represent

severe ulcerative colitis, patients who had run the gamut of conventional treatments without the slightest improvement. The results of authors' treatments appear startling.

"Therapeutic sleep" is the authors' expression, they use in explaining their method to the patients. The hypnotic sessions, in the beginning, are given daily, later on several times weekly and/or p.r.n. The time up to complete recovery varies from 2-10 months, including treatments of occasional relapses. The authors publish x-rays showing all the changes of ulcerative colitis before the treatments and pictures after successful conclusion, presenting nearly normal appearance of the colon. The sigmoidoscopic findings after the termination of the treatments are not given.

In 1942 the authors published reports of success with their treatment of ulcerative colitis. Still the number of cases is too small to draw any final conclusions.

The authors think it worthwhile to subject a large number of patients to hypnotic treatments, especially before any surgery is contemplated. In their opinion many a patient with a grave prognosis could so be cured and rehabilitated.

HANS J. JOSEPH

LIVER AND BILIARY TRACT

LIVER FUNCTION TESTS: Louis S. Smith and Joseph H. Gast. Texas J. Med. 51:67 (Feb), 1955.

The differential diagnosis in liver disease rests on a complete history, physical examination and carefully chosen liver function tests.

Screening tests will show dysfunction of 1) pigment metabolism—serum bilirubin test; 2) excretory mechanism—Bromsulfalein retention; 3) protein and lipoid metabolism—thymol turbidity and cephalin flocculation tests.

Bilirubin or serum bile tests show presence or absence of jaundice and should replace the icterus index tests which are often contaminated by food or drug "yellows" in the serum. The van den Bergh test is often misleading clinically and can easily be replaced by the bilirubin test with the urobilinogen determinations. A bilirubin test showing 1.0 mg. should be investigated, since frank jaundice does not appear until the concentration is 2.5 mg. per 100 c.c.

Bromsulfalein is particularly sensitive in large doses and a short reading time (5.0 mg. per kilo read at 30 minutes after injection). In this test the dye must reach the liver in a normal circulation time; cardiac failure will slow circulation and give false positives.

The thymol turbidity test depends on the release of lipoproteins from damaged liver cells and should be taken on fasting blood to avoid nonhepatic sources of lipoproteins. The test reflects liver cell damage and is important in infectious hepatitis, early cirrhosis and liver necrosis and also in late extrahepatic obstruction.

The cephalin-cholesterol flocculation test, similar to the above, requires 24 hours to perform, but is a superior determination when read on the photometer. It becomes abnormal earlier than any other test in cell damage except in infectious and serum hepatitis.

Urine urobilinogen differentiates between hepatic and nonhepatic icterus, high values being found in anemias of hemolytic origin, early infectious hepatitis and acute liver necrosis. It is useless in abnormal renal retentions and high renal thresholds.

J. EDWARD BROWN

ERFAHRUNGEN MIT DEM GALLENKONTRASTMITTEL "TELEPAQUE": Th. Meese and Th. Lind. Deutsche med. Wchnshr. 80:175 (4 Feb.), 1955.

Oral cholecystography is technically simple and shows little toxicity but requires 10-14 hours to opacify the gallbladder and depends on full cooperation of the patient while the intravenous method yields an x-ray after two hours and does not require a reliable patient; however, it is more dangerous.

Fifty patients received Biliselectan (Priodax), another 50 were given Telepaque. If no gallbladder shadow was obtained with the oral methods Biligrafin (Cholografin)

was injected intravenously. Telepaque showed a greater contrast density allowing visualization of several calculous and noncalculous gallbladders not demonstrated with Biliselectan. In quite a few cases Telepaque even opacified the bile ducts. Residues of contrast material in the colon were more frequently observed after Telepaque but did not interfere with the x-ray interpretation. Opacification of the gallbladder persisted longer after Telepaque than after Biliselectan. Toxic reactions were mild after either oral preparation, but occurred less frequently with Telepaque. The greater density of Telepaque might occasionally obscure a faint stone shadow on the routine view, but as this dye allows fairly good visualization under the fluoroscope, spot films with graduated compression can be made. Only one-third of all gallbladders not visible with Telepaque could be demonstrated after intravenous Biligrafin.

H. B. EISENSTADT

ERRORS IN THE DIAGNOSIS AND TREATMENT OF LESIONS OF THE BILIARY TRACT: Waltman Walters. Mississippi Valley M. J. 77:57 (Mar.), 1955.

Patients with a history of cholecystectomy and with symptoms of biliary colic may have stones in the common duct or the extrahepatic bile ducts, which may be found on intravenous cholecystography with Cholografin. Most cases of postcholecystectomy pain are due to retained stones or stones in the common or hepatic bile ducts or the stump of the cystic duct, to pancreatitis, or to spasm or contracture of the sphincter of Oddi. When stones are not present in the common duct, about 70 to 75 per cent of these patients are relieved of symptoms for a year or more by either sphincterotomy or choledochoduodenostomy.

Failure to recognize the possibility of cholecystic disease is not uncommon. Frequently, the pain associated with gallstones is not alone pain due to obstruction of the cystic duct, but is due also to associated gastrospasm or pylorospasm. As a result of gastrospasm, the patients with cholecystic disease will have an ulcer-like type of distress. Pain in the left upper abdominal quadrant may be caused by a diseased gallbladder or by inflammation in the pancreas, which latter may be the result of interference with emptying of the pancreatic duct due to a stone in the ampulla. In some cases of this type, the pain may be precordial in location, leading to the erroneous diagnosis of angina pectoris or coronary thrombosis. It would be advisable in all routine physicals on patients who are more than 45 years of age, especially women who complain of bizarre types of dyspepsia, to do cholecystograms.

The presence of a single large shadow in a cholecystogram of a seemingly single stone does not exclude the possibility that multiple small stones also may be present, some of which may be obstructing the cystic or the common bile ducts. The presence of "only mild symptoms" should not contraindicate cholecystectomy, as it is better to remove a gallbladder before its continual insults to the pancreas and liver produce chronic infection in them. Gallstones are always associated with some degree of infection, and usually produce obstructive symptoms and enter the common bile duct at some time or other. Involvement of the liver or pancreas increases the risk of an operation and decreases the likelihood of relief of symptoms.

Tests of liver function in the presence of jaundice are not diagnostic of themselves as to whether the jaundice is obstructive or due to disturbances within the liver. The various flocculation tests do help, and are low in cases of obstructive jaundice and cases of intrahepatic jaundice. In the former, the alkaline phosphatase is increased, and is normal in the latter.

During operation, every enlarged common bile duct should be opened and its interior carefully explored with scoops for stones. It is advisable to ascertain that an ampullary lesion or a carcinoma of the pancreas is not present in any patient who has had evidence of obstructive jaundice, and it is deemed advisable for the surgeon to open the duodenum to examine the ampulla. In patients whose common bile duct is opened and explored and a T tube placed in it for temporary drainage, it should be axiomatic that a cholangiogram should be done to demonstrate that the duct is unobstructed before the T tube is removed.

ARNOLD L. BERGER

BOOK REVIEWS FOR GASTROENTEROLOGISTS

X-RAY ATLAS AND MANUAL OF ESOPHAGUS, STOMACH AND DUODENUM: Dr. T. J. J. H. Meuwissen. 687 pages. Elsevier Press, Houston, Texas. Price $25.00.

This atlas which is a compilation of two and a half decades of dedicated work, looms as a very important contribution to the allied fields of radiology, gastroenterology and internal medicine in general.

Containing one of the most exceptional collections of x-ray films ever reproduced in a textbook, the clarity and quality are even more enhanced by utilizing cream colored mats as a background.

Based upon years of experience, correlating clinical findings with roentgenograms, Dr. Meuwissen has produced along with the very excellent translator, a volume of which he can be justly proud.

The atlas is divided into four general sections, complete with text, line drawings, normal and pathological x-ray films, each of which was the author's case. Complete, concise case histories including all pertinent x-ray findings accompany most of the roentgenograms.

The contents include esophagus, stomach, duodenum and postoperative stomach. Two exceptionally well developed subgroupings include gastric ulcer and gastric carcinoma, wherein the author again points out how questionable gastric analysis is in the presence of benign ulcer or malignancy. The use of the sedimentation rate as a true guide to degree of malignancy also has pitfalls in the author's opinion.

Because there is enough of a basic approach to each subject, it can be utilized by the medical student and practitioner as a most welcome adjunct to the usual texts. Speaking for the general practitioner as well as the radiologist and gastroenterologist, we should be grateful to the author for the opportunity of adding this to our list of "must read".

THE MACMILLAN HANDBOOK OF ENGLISH: John M. Kierzek. 579 pages. The Macmillan Co., New York, N. Y., 1954. Price $3.00.

This book is primarily a college book written for classroom instruction, but should not be confused with the ordinary books dealing with rhetoric. Physicians preparing papers for publication or to be read at medical meetings, will find many useful suggestions in this book.

The reviewer recommends it highly for those physicians who are contemplating writing scientific papers.

ANGIOPATHIA DIABETICA—KONSERVATIVE BEHANDLUNG DES ZUCKERBRANDES: Prof. Dr. M. Burger, Leipzig. 190 pages, 81 illustrations. Georg Thieme Verlag, Stuttgart, 1954. Price $16.40.

A well written and documented bok dealing with the pathology of diabetes and the prevention of operative interference when possible, by the use of insulin, sulfa and antibiotic therapy.

Unfortunately, the book will find a limited sale among the general practitioners and even the internists, because of its high technical calibre and language barrier. It is the suggestion of the reviewer that the publishers make every effort to have this very useful volume translated into various languages.

1954-1955 YEAR BOOK OF GENERAL SURGERY: Edited by Evarts A. Graham, M.D., with a section on Anesthesia edited by Stuart C. Cullen, M.D. 500 pages, illustrated. The Year Book Publishers, Inc., Chicago, Ill. Price $6.00.

On page 15, the reader will find an interesting abstract dealing with porphyria in surgery. A simple diagnostic test was made by placing the acidified urine in the sunlight. The urine changed from a yellow to a pink or port wine color and this was diagnostic of porphobilinogen, signifying impaired liver function which was verified

at autopsy. A more elaborate test may be made by the Watson-Schwartz test.

Page 33, deals with low sodium syndromes of surgery, outline for practical management, should be carefully read by the intern, resident and surgeon. Another pertinent subject on page 37 deals with safe parenteral fluid therapy. Intravenous alcohol therapy as a pain reliever after surgery used as a morphine substitute, is very interesting and instructive (page 41).

For the general surgeon and the internist, "The Abdomen in General"—page 247, etc., will be interesting review. Intravenous cholangiography by the use of biligrafin and radio solution are the newer diagnostic procedures for visualization of the biliary apparatus. Contraindications for use of these preparations must be taken into account.

The reviewer recommends the Year Book on General Surgery, as well as all the other Year Books, as excellent means for keeping up with the latest development in the medical field.

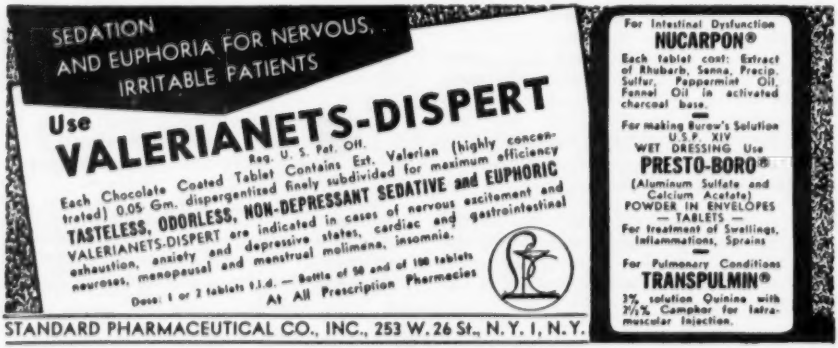
MANAGEMENT OF ADDICTIONS: Edited by Edward Podolsky, M.D., Dept. of Psychiatry, Kings County Hospital, Brooklyn, N. Y. 413 pages. Philosophical Library, New York, N. Y. Price $7.50.

Here is a book that should be read by all medical men, whether general practitioners, specialists or surgeons. The list of contributors are either neurologists, psychiatrists or internists who have devoted considerable time and effort to make this volume a *vade mecum* for the treatment of addicts to alcohol and various drugs.

The use of adrenal steroids and ACTH in the treatment of alcoholism on page 143 is well written. On page 347, drug addictions are discussed by Arova and Sharma. Stimulants—cocaine, benzedrine, opiates and synthetic depressants; analgesics like methadone, demerol, hypnotic and sedative drugs like chloral, paraldehyde, bromide, barbiturates, alcohol and cannabis.

The treatment of drug addictions by Williams on page 358 and Schlomer's chapter—page 370, discusses morphine withdrawal in addicts, while Vaisberg, page 377, advises the use of benadryl in the narcotic withdrawal syndrome and in other conditions.

All in all, Dr. Podolsky should be congratulated for bringing these things to the attention of the medical profession, social workers and others interested in the prevention and rehabilitation of these addicts.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Nausea and vomiting, hiccups, pylorospasm	104	80%
Hiatal hernia, gastro-duodenitis, upper gastrointestinal bleeding	15	87%
Gastritis medicamentosa	8	100%
Genito-urinary disorders	23	74%
Postoperative nausea and vomiting	15	90%
Nausea and vomiting of pregnancy	7	71%
Gall bladder disorders	10	80%

*In many cases, partial relief was obtained

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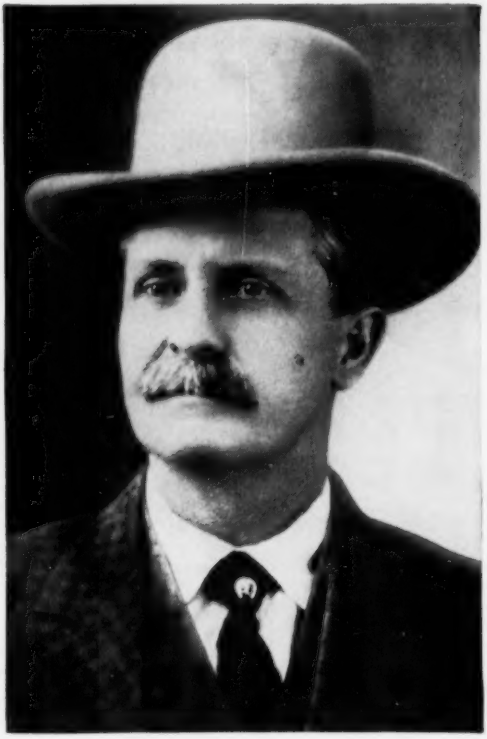

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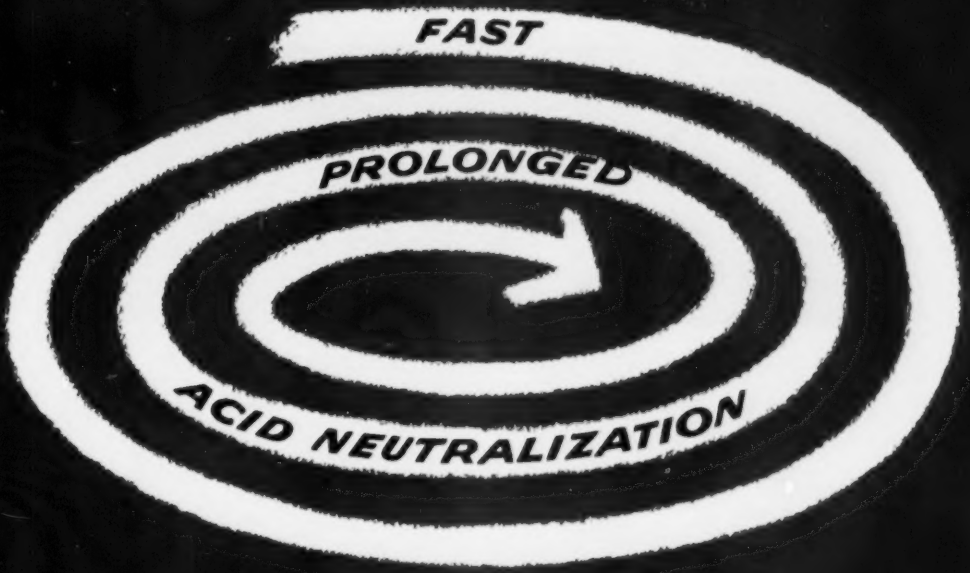
*Steigmann, F., and Goldberg, E.: Ambulatory Continuous Drip Method in the Treatment of Peptic Ulcer, Am. J. Digest. Dis. 22:67 (Mar.) 1955.

†Mg trisilicate 3.5 gr.; Ca carbonate 2.0 gr.; Mg oxide 2.0 gr.; Mg carbonate 0.5 gr.



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1. Rosett, H.S., and others: Ann. Int. Med. 36:98 (Jan.) 1952.
2. Jankelson, I.R.: Am. J. Digest. Dis. 14:31 (Jan.) 1947.

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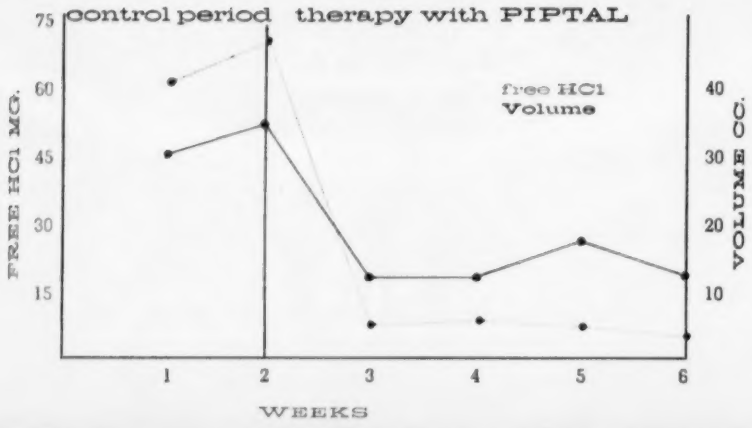

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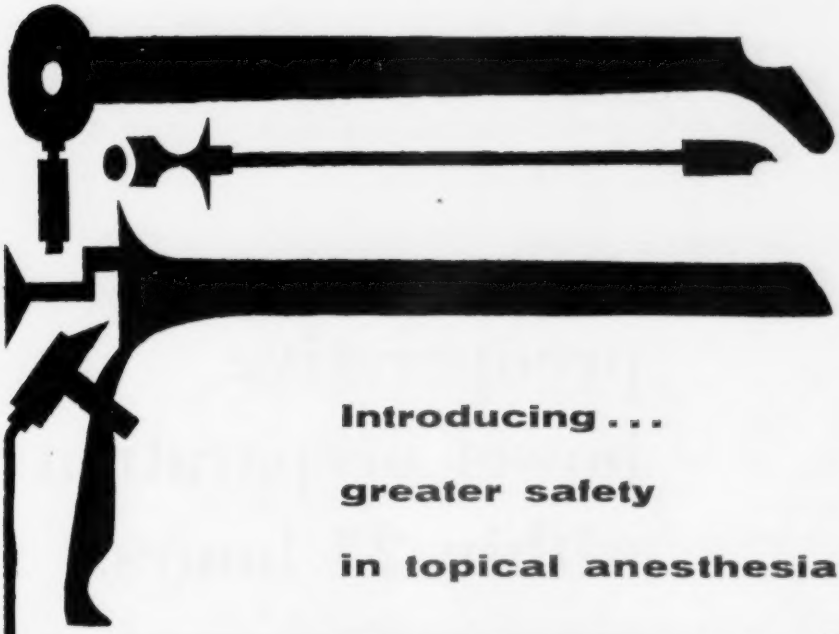

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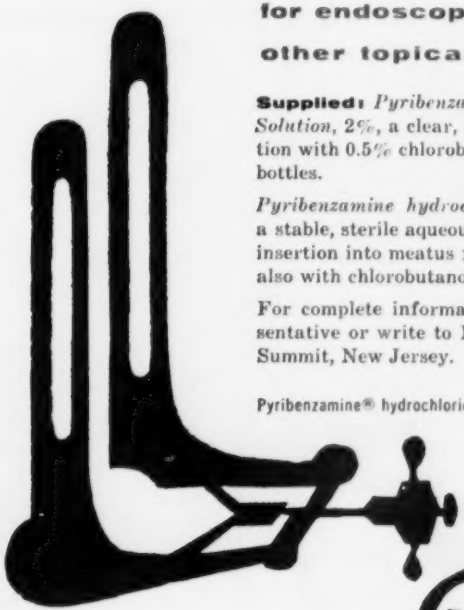

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